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KIDNEY LESIONS PRODUCED BY TISSUE BREAKDOWN (CYTOST). PATHOGENESIS AND TREATMENT.*

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INTRODUCTION.

Since biology has now become a part of surgical science much light has been shed upon obscure pathological conditions which daily confront the surgeon.

Injuries due to disease, to accidents and those associated with operative procedures still harrass the surgeon. This is because of the mysterious clinical happenings which burst forth unexpectedly.

Cushing[1] in his presidential address before the October, 1922, meeting of the American College of Surgeons said: "Any one of us may at any time acquire an utterly erroneous impression regarding a pathological lesion and the effectiveness of our operative procedure in a given case," and continues, "but knowledge to be exact must be based on something more than operating-room and bedside impressions."

By a better knowledge of modern biological principles as distinguished from clinical pathology these surprises will not occur. In addition to mechanical skill and pathological findings we must go deeper into the cell itself. The results of the modern biological investigation are presented showing its clinical significance in surgical practice.

PART I—TRAUMA

One of the earliest known forms of trauma is the burn. Human tissue that has undergone the charring action of fire, of whatever kind, flame, steam, *x*-rays, diathermy, chemicals, etc., when absorbed into the organism, is highly toxic. Death from burns is due, as has long been recognized, to the toxic action of the burned tissue, producing shock in the organism.

Whether this burned tissue is produced in vivo and absorbed, or is charred outside the body and injected, is immaterial. It produces the same effect, the result of which is death.[59, 60, 112, 120]

The constant pathological findings are general splanchnic congestion and kidney lesions. Instead of studying the effects of kidney lesions on the organisms, it is far more important for the surgeons to know what effect the organism has upon the kidneys. The kidney changes may be used as an index of tissue toxemia. The discovery of the biological and pathological effect of homologous tissue extract, supplies the foundation on which practical surgery and internal medicine rest.

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After following the toxic effects of damaged tissue from burns it will not be so difficult to trace the effect of damaged tissue from other causes, physical, chemical or mechanical. Autolysis or tissue breakdown from local or general inanition or other causes, can be traced from the time the tissue toxin is liberated by the injury to the final pathological effects in the organism.

The "dosage" of the homologous tissue toxin not only determines the degree of the pathological effects, but the quantitative variation of the antitoxin or immunity of the organism to the tissue toxin defines the character of the disease in both the acute and chronic stages.

History of Kidney Lesion. The study of the initiatory steps in acute and chronic kidney disease, nephritis or Bright's diseases, has been followed to a very early stage of development, especially in surgical cases, without finding its real beginnings. This has left room for much speculation.

A review of our text-books and of the many brochures on kidney complications will reveal the innumerable theories that clutter medical literature.

At the Association Francaise d'Urologie, at Strasbourg, October, 1921, I presented the results of many years of research in which kidney lesions gave an index of the degree of tissue toxemia. A further contribution was presented before the Paris meeting of the Association, October, 1922, showing the effects of burned tissue and of autolysis in producing "constitutional disease" with kidney lesions.[124, 125]

The Effect of Burns. The clinical interest in the etiology of kidney disease has been much stimulated by post-mortem studies made in surgical cases of severe burns. The immediate effect of burned tissue on the kidneys, resulting in death within a few hours, indicates a direct toxemia. At best, clinical work can furnish only an hypothesis for the pathogenesis of kidney disease. A general agreement has been made by several clinicians after a post-mortem study of kidney lesions, resulting from burns.

In 1897 Bardeen[2] reported necropsy findings in five fatal cases of burns. He found degenerations of the kidney and suggested that some circulatory poison produced these lesions and that the toxemia may be caused by alterations in blood and tissues.

Pfeiffer[4] (1905) after a review of the literature and clinical study and post-mortem findings, arrives at the same theoretical conclusion as Bardeen.

Similar clinical pathological studies were made by Eyff[6], Vogt[7] and more recently by Weiskotten[8], who also asserts that the kidney lesions and other lesions following burns, are due to a specific poison in the circulating blood. Becky and Schmitz[9] assert the presence of protein of unidentified character in the urine of patients after severe burns. In Pfeiffer's[5] more recent clinical pathological findings, he shows that scalding is only a special type of pathological

condition from the action of heat. The cause of the kidney lesions is assumed to be the autointoxication of the products of protein metabolism, characteristic of the whole group. This Pffeiffer asserts causes the breakdown of the physiological defensive system.

"This will", Pfeiffer further states, "explain many hitherto obscure pathological processes besides those from burns". These statements show how the theories gleaned from empirical clinical observations often agree with the more exact conclusions from experimental research work.

Burns produced by "actual" cautery or electric cautery, resulted in the same effect. Warthem[11], describes chronic kidney fibrosis following the repeated exposure of the peripheral tissues to the *x*-ray.

Burns by sun, *x*-ray, radium, diathermy, vary in their effect according to the intensity of the burn and the character of the tissue cells that are exposed. My researches for the past 25 years have been devoted to effects on organisms of tissue damaged by various mechanical, physical and chemical agents. It is not the agents that produce the immediate biological effect, but the product of the damaged tissue that is released by any of the mechanical, physical or chemical agents.

Results of Experimental Research. By experimental research on animals with tissue toxin and antitoxin, in the last 25 years the writer has established definite scientific results which compare well with clinical experience. The practical application is made available by the knowledge of the reaction between tissue toxin and antitoxin. (See bibliography.)

When it was learned that burns produced kidney lesions, and that these resulted directly from the burned tissue substance, it was most natural to seek the poison in the tissues. Various chemical substances of tissue origin have been regarded as the toxin that produced the kidney lesions, such as foreign protein, metabolic products called "metabolites," lactic acid, CO_2, amino acids, histamines, tyramine, etc.

Injection of a solution of burned tissue, produced much the same symptoms and pathology as severe clinical burns. When burned tissue was burned to a crisp, charred to a cinder so no organic matter remained, a watery solution of this burned tissue ash was injected into animals. This caused death from shock with kidney lesions. All organic matter, foreign proteins, amino acids, histamines, etc., were destroyed, hence we came to the conclusion that we were dealing with a non-protein toxin that was found in the incinerated ash. This was in harmony with clinical experience in severe burns with tissues charred to a cinder.

This discovery is of stupendous importance to every surgeon, for it explains, as Pfeiffer intimated, so many obscure pathological processes other than from burns.

Clinical Observations Compared with Experimental Burns in Animals. Animals suffering from accidental burns showed similar symptoms and pathology to those observed in human individuals. They both suffer from splanchnic congestion and kidney lesions. It is remarkable that very severe burned tissue, charred to a crisp cinder, may show no symptoms in man or animals, and yet they die within a short period. The marked splanchnic congestion and advanced kidney lesions indicated the severe toxemia that had occurred.

Hunt[16] noted this lack of painful symptoms after the most severe burns, where the tissues are charred, and states, "the intelligence takes no cognizance of the vast calamity. I have seen, after deep and extensive burns, what seemed to be a living soul looking out of a crisp, charred and dead body. I have seen a child thus burned playing with toys an hour before its death."

When the tissues are burned completely, charred to a cinder, the nerve endings are also destroyed. This does not, however, prevent the absorption of the incinerated tissues which causes the toxemia and death. In fact, it seems that tissues that are burned to a crisp, charred to a cinder, appear most toxic because the organic substances acting as a "buffer" are destroyed. This was made perfectly clear by the following experiment.

A portion of the burned animal's tissue which had been charred to a cinder was pulverized in a mortar and water, definitely measured, was added. This incinerated tissue substance was injected into normal animals of the same species. This caused shock and death of the animals injected. This was the same as occurred with burned animals. Post-mortem findings were splanchnic congestion and kidney lesions. To make sure that it was the incinerated tissue ash and not the albumins or protein that was the toxin, fresh animal tissue was weighed and placed in a crucible and burned at 300° C. to a white ash. To 1 part of this ash (weighed) was added 10 parts of water. Injecting 1 cc. of this tissue ash solution into animals of the same species from which the tissue had been taken caused death from shock with kidney lesions.

One point was made decisive, that general "toxemia" caused the kidney lesions. This toxemia does not, however, result from the organic substances as we had previously supposed. Clinicians have wondered why charred tissue was so toxic and also why some individuals are more susceptible to the toxin than others. I made a post-mortem and histological study of the case of a child that died within a few hours from a severe burn of the thigh. The burned area was not large, but the tissues were charred to a cinder. The extent of the injury showed no evident relation to the degree of shock symptoms and pathological changes. Some individuals are very resistent while others are extremely susceptible to even slight injury. This was found as a constant observation in all our experiments on shock from burns or wounds.

Bosquette[18] in his discussion on soldiers shocked from wounds, burns, etc., stated that "It seems as if there was some individual predisposition to shock as an *intangible* factor common to so many unlike cases. There is no evident relation between the type of wound and shock."

This unexpected difference in susceptibility to the effects of damaged tissue is one of surprise to surgeons the world over, as will be seen in the literature. Apparently *occult* kidney lesions have arisen from very slight injuries of the body. Experimenters for years have reported this variation in animals' susceptibility to shock from various types of

injuries, and the kidney lesions have seemed a natural sequence of the lack of natural resistance or immunity.

This individual variation in natural immunity to their own tissue toxin has been a clinical but empirical fact observed for ages. Now the experimental work which parallels the clinical observations raises this out of empiricism by the light of scientific research with quantitative determination. This variation of immunity to an animal's own toxic tissue substance, when released in the organism, gave the best evidence of the nature and the fact that *the organism's own tissue substance is the toxin.* Decreasing natural immunity or increasing natural immunity, causes the variations in the toxic symptoms and pathological findings.

These facts have explained the hitherto obscure surgical pathological processes that have been veiled under the name of "surgical risks." As will be seen, when practically applied in clinical, compared with the experimental work, we are concerned with a specific toxin. This is only relative to the specific antitoxin, the natural immune body of every organism. It is the degree of this immunity which fluctuates and causes the variations of normal and pathological processes.

At best we must admit that clinical observations may be used only as illustrations of scientific experimental findings.

Clinical observations alone cannot be accurately measured and controlled. Nor can repeated laboratory observations be made and modified as in animal experiments, or the actual conditions seen in nature be reproduced.

Experiments. The reason for the interest taken by both biologists and clinicians in experimental burns on animals is that many obscure etiological facts have been uncovered. The immediate destruction of tissue was constantly followed by a general reaction in the organism, with always the same pathological findings in the splanchnic zone, including kidney lesions. A burn, and then death; and the burned ash injected in a normal animal, was followed by prompt death with the same pathology as the burned animals. This left nothing for speculation as to the etiology. Tissue damaged from other causes produced precisely the same effects as burns. Tissue disintegration and the toxin balanced by the tissue antitoxin, created a new world of biology applied to surgery and medicine.

Method.—In experimental burns under general anesthesia, some of the symptoms that arise, such as shock, are produced in part by the anesthetic (1903)[73]. Local anesthesia with novocaine, will accomplish the purpose without the complication of added shock. Branding irons were heated to a white heat to burn the tissues to a charred crisp-like cinder. Heated clamps were used with the tissues protected below the burned area. The complete destruction of the tissue, including the nerve endings, prevented any secondary pain, and the so-called "reflex action" need not be assumed. The animals passed into shock gradually, and painlessly. When death occurred it was without any marked symptoms except the fall in blood pressure, temperature, etc., with the usual shock symptoms. These observations parallel clinical experience with severe burns, when tissues are charred to a crisp.

When using hot irons or clamps, they were brought to a white heat to cause complete charring of the tissues. Part of the charred crisp tissue residue was pulverized in a mortar, suspended in water and injected into normal animals. This charred tissue ash solution injected in animals, caused death from shock with kidney lesions, the same as the direct effect of burns in the animals.[59 60 112]

In the following table the results only are shown. The burns were made on the back and buttocks. Muscle and skin were grasped and burned with irons at "white heat". The time required was but a few moments before the charring of the tissues was complete. If the iron was reduced to dull heat, it was replaced. The time after the burn, before general symptoms were apparent, varied in different animals. Usually within two hours the shock symptoms were manifested with the usual fall in temperature and blood pressure.

As these symptomatic shock changes have been described many times by me (see bibliography) it will be necessary here only to refer to the pathological changes that were found post-mortem. As will be seen by the table, the time of death varied in different animals from six to twenty-four hours after the burns. Animals that survived indicated a higher degree of natural immunity.

When we refer to natural immunity in man or animal, we know that it varies in different individuals and also we have learned that the degree of immunity is relative to the degree of the toxic substance or the toxin of the animal's own tissue substance. It was the main phenomenon of individual variation of immunity that led to the discovery of natural immunity to tissue toxin. It was, therefore, but a short step to devise methods to increase the animal's antitoxin against their own tissue toxin. The method of producing active and passive immunity was evidently not generally realized, and was regarded as "intangible" until experimental and clinical work had established the principle on a sound scientific basis. (See bibliography.) The finding of the antitoxin immunity also further established the principle of the action of the tissue toxin, as the etiology of disease processes.

The following tables show: *first,* the effect of the burns produced; *second,* the charred tissues removed, and a solution of the tissue ash injected into homologous animals:

No.	*Animal*	*Onset of Symptoms*	*Death*
1	Cat	2 hrs.	8 hrs.
2	Cat	1½ hrs.	10 hrs.
3	Cat	4 hrs.	24 hrs.
4	Cat	3 hrs.	63 hrs.
5	Cat	5 hrs.	Recovered
6	Dog	4 hrs.	24 hrs.
7	Dog	2 hrs.	10 hrs.
8	Dog	3½ hrs.	22 hrs.
9	Dog	2 hrs.	8 days
10	Rabbit	1½ hrs.	6 hrs.
11	Rabbit	2 hrs.	7 hrs.
12	Rabbit	4 hrs.	5 days
13	Rabbit	2 hrs.	Recovered
14	Rabbit	3 hrs.	5 days

The crisp charred tissue was removed from the burned animals, pulverized, and 10 cc. water added to one gram of the ash. One cc. was injected intravenously into cats and rabbits, 2 cc. into dogs. These were injected into homologous animals.

No.	*Animal*	*Onset of Symptoms*	*Death*
15	Cat	Immediately	
16	Cat	Immediately	
17	Cat	Five minutes	2 hours
18	Cat	10 minutes	Recovery
19	Cat	½ hour	24 hours

No.	Animal	Onset of Symptoms	Death
20	Dog	Immediately	
21	Dog	10 minutes	5 hours
22	Dog	½ hour	10 minutes
23	Dog	Immediately	½ hour
24	Rabbit	Immediately	
25	Rabbit	5 minutes	6 hours
26	Rabbit	4 minutes	Recovery
27	Rabbit	5 minutes	12 hours
28	Rabbit	10 minutes	Recovery

Pathological Findings. Inasmuch as this is to be a report confined to lesions of the kidney, it must be remembered that the reaction is a general reaction, and that all kidney lesions are usually only the renal expression of a "constitutional disease," of a general toxemia. This has been suspected for years, but only by experimental work has it been fully demonstrated.

Within five minutes after the injection of the tissue toxin (cytost), kidney lesions appear, usually in the form of a glomerulitis. The approximal tubules later become involved.

Burned Animals. The degree of the burn and the time between the burn and death, will vary somewhat the pathological picture found in the kidneys. Both the degenerative type and inflammatory, proliferative type occur in the glomeruli and approximal tubules. The degeneration with blood stasis causes further tissue breakdown. This necrosed tissue causes secondary changes in the kidney tissues. If it continues, the cells multiply in great numbers, which form the chronic proliferation, and may thus constitute irreparable damage to the kidney. Acute glomerulitis is probably always due to this tissue toxin, as it appears within five minutes after injection of the tissue toxin. The animals that did not die from burns did not fully recover from their kidney lesions. Degeneration continued with secondary proliferation. When injection of burned tissue was made and the animals recovered, similar secondary lesions were also found in the kidneys.[112]

General Pathological Findings Associated with the Kidneys. It must be remembered that liver and lung lesions also appear in these general tissue toxemias, occurring either from burns or from the injection of burned tissues. The liver and lungs may "clear up." The absorption of cellular products may leave but comparatively little evidence of the great damage that had occurred. The liver and lungs are supplied with two "sets" or double vascular blood supply, as the liver "functional" (portal vessels) and the "nutritive" (hepatic artery). The kidney depends chiefly on the renal vessels with some auxiliary anastomosis. This single "functional" blood supply is severely taxed in these general tissue toxemias. When blood stasis occurs in the kidney, especially of the glomeruli and the approximal tubule region, recovery from damage is slow and secondary degeneration takes place. This causes further tissue breakdown with secondary reaction of the proliferative type which is responsible for the permanent kidney damage.

The Effect of the Removal of the Burned Area. The above experiments harmonize with clinical experience, viz., that tissue burned to an ash is highly toxic; and the injection of a solution of the tissue ash into a normal animal of the same species, caused death with kidney lesions. This proves that the toxin is not a "foreign protein," a product of metabolism or amino acid, histamine, etc.

The normal tissue removed from the body and burned to a white ash in a crucible become highly toxic and specific to the species, as will be seen in the protocols here, as in other publications.[106, 112, 120] This tissue ash, which is so highly toxic, caused the symptoms and death. Therefore, the removal of the burned area before the toxin (like snake venom)[34, 43, 58] had permeated the organism, ought to save life. When burned tissue was removed at once from the animals and with it all damaged tissue, no shock symptoms followed and death was averted.[112] (See Protocols, VIII). The burned tissue that had been removed was transplanted to a normal animal of the same species. Shock symptoms developed in the second animal receiving the burned graft which ended in death. The burned animal from which the graft was removed recovered with but slight shock symptoms.[112]

These experiments correspond to the conditions found after injuries from wounds in the war. The removal of the damaged tissue ("épluchage" or, as the Americans and English applied the term, "débridement") when done early—before the profound effects of tissue toxin had produced profound shock—saved life. The damaged tissue taken from the mangled leg of a cat was inserted or transplanted within the muscle substance of the leg of a normal cat. The absorption of this tissue toxin caused gradually deepening, profound shock, and finally death in sixty-four hours. The post-mortem examination revealed subacute reaction in liver and upper alimentary tract. The kidneys showed hemorrhages with marked congestion in the glomerular region, also venous stasis with clumping of the corpuscles in the region of the straight tubules. (See figures 9, 10, 11, 12, and 13 in reference 112.)

The Practical Application of the Principle. The removal of damaged tissue, ("débridement") before the tissue toxin had produced its fatal effect, saved life. Major Lemaitre[12] was one of the first to adopt Larrey's method which was, according to Senn,[13] the removal of damaged tissue to prevent the effect "commotion" (shock). Lemaitre claimed that excision of damaged tissue gave opportunity for the body to regain its natural immunity. It was not as others had conjectured, to stop infection, as that was ignored. As Lemaitre stated "a temporary return to faulty pre-war methods with antiseptics, led to the prompt reappearance of the high mortality."

As Lexer stated in the last German Surgical Congress,[14] "general toxemia arises from so-called tissue toxins. An example is the toxins associated with

burns," and, further, "shock may be explained by intoxication through decomposition products," and he concludes that "tissue toxins alone play an important rôle." As he explains, "they break down the defensive measures of the organisms." The importance of increasing the antitoxin against tissue toxin from damaged tissue is emphasized now by clinicians as well as experimental biologists.

Henri Hartmann, before the French Academy of Medicine, January, 1922,[129] previous to Lexer's paper before the German Congress, showed not only that general toxemia arises from tissue toxins, but he gave a detailed review of my work of over 100 publications before the war. He showed that the priority of this research work, now generally accepted, belongs to America and quoted Munch's report[128] as further evidence that the toxemia of tissue breakdown in producing disease was established in this country. Reference was also made to the brochure of G. Jeanneney of the Université de Bordeaux on "Choc Traumatique"[126] in which he likewise reviewed my work, arriving at the same conclusion.

Summary of the Effect of Burned Tissue. In the experiments two important facts are shown that are in harmony with clinical experience:

(1) Burned tissue, burned to an ash, is highly toxic to the organism when it is allowed to remain in the organism.

(2) The toxic character of the tissue ash is proven by removal of the crisp, charred tissue, and injection of a solution into a normal animal of the same species. This caused general shock symptoms and death with kidney lesions.

(3) Further, the saving of life by the removal of the cause, which is the damaged tissue, gives additional evidence of the etiology of tissue toxemia in producing the general symptoms and the kidney lesions.

Scalds. Scalds from hot fluids it is known, as Pfeiffer also mentions, produce injury similar to burns from dry heat. The effect is dependent upon the intensity and duration of the application. Death from scalds is due to the tissue toxin of damaged tissues just as dry heat releases the tissue toxin from the tissues. It was found in my experiments as early as 1896,[34] that the toxin derived from the fasting stomach may be heated from 53° to 100° C. and still retain the toxic character. The coagulated material was removed by filtration through a Pasteur filter. This yielded a filtrate that was toxic on injecting the residue into animals.

The scald may destroy tissue but does not reduce it to an ash, but the same toxin that is found in the ash of burned tissue is also found in the tissues from scalds. Moist heat just sufficient to cause the extrusion of the cellular toxin from the living cells, will produce similar toxic effects and death as burns from tissues charred to an ash.

This poison released by moist heat is, therefore, not protein or anything that has been named by clinical physiological chemists. As will be made more definite and clear, other agents than moist or dry heat will cause the same tissue toxin to escape from the cells and result in general toxemia.

We know that tissues vary in their resistance to the effects of burns and all cauterizing agents. Thus the skin is more resistant than the mucous membrane, and the peritoneum, so that we would expect a degree of temperature that would have no damaging effect upon the skin might produce great damage to the peritoneum. The skin and the peritoneum vary in their resistance to burns and many similar agents. This is shown in the following experiments.[60, 59]

Experiment No. 15.—"Dog under anesthetic. Heat introduced into the stomach at 65° C., which for the first ten minutes caused contraction of the splanchnic vessels. In 15 minutes there was observed marked dilatation and at the end of 20 minutes venous engorgement, with marked hyperemia of the splanchnic area. This was followed by rapid heart-beat with complete collapse at the end of 30 minutes."

Experiment No. 16.[60] "Dog under anesthetic. Water injected into the colon at 68° C. This was continued for 30 minutes. Marked splanchnic congestion followed by collapse. The animal recovered from the anesthesia, but died next day. Observations showed a sloughing of the mucosa of the colon.

Experiment No. 28.[59] "Dog; the abdomen was opened and hot water placed in the abdominal cavity for 1 hour and 20 minutes. The temperature of the hot water bags was 58° C. Result: animal died within 24 hours. Autopsy showed blebs on the intestines resulting from the continued application of hot water bags at 58° C."

As the writer concluded from a series of similar experiments "the degree of the temperature to produce desiccation of the tissue—a burn—varies in different locations of the body. This is clearly shown by the application of heat within the stomach at 65° C., within the colon at 68° C., and within the peritoneal cavity at 58° C. The length of time of the application of the heat is also a factor in causing cell necrosis and the liberation of the "shock toxin." Some cells more readily release their contents than other cells. Blebs formed as a result of heat applications at 58° C. to the peritoneum. So delicate is the peritoneum that even exposure to air and handling the tissue acts similarly to a burn. The peritoneal fluid after the exposure to air contains necrosed cells. On injection of this peritoneal fluid I caused shock and death to normal animals of the same species.[69] The shock of the viscera exposed to air is not only of scientific biological importance in all operations but is of a great practical significance not yet fully appreciated by surgeons. Too much attention to mechanical technique rather than biological principles eclipses the vision of the fundamental biological law with which they are much concerned. Hence the surgeon frequently unwittingly causes grave pathological lesions and death by the operation. Kidney lesions may follow the direct effect of the surgical procedure.

Summary of Effects of Burns which Liberate the Toxic Cell Contents. In all our researches we have shown that in kidney disease a constitutional reaction must take place first, before the kidney becomes involved. It may be looked upon as an intimate

part of a constitutional reaction against the homologous tissue toxin, hence we take up first the most common and accepted agent, the burn, which releases the toxin in the organism. Then we may proceed to the other agents that cause the tissue toxemia with kidney lesions. This leads us to the next group of toxemias produced from causes other than burns.

PART II—CAUSES OTHER THAN BURNS WHICH RELEASE THE CELL CONTENTS.

INTOXICATIONS FROM THE ALIMENTARY TRACT AND GENERAL METABOLIC DISORDERS.

Surgeons have discovered, especially in abdominal operations, that the most obscure and perplexing phenomena are the intoxications arising from the alimentary tract associated with general metabolic disturbance.

Often unexpectedly, as if led into ambush, surgeons experience in certain cases alarming gastro-intestinal symptoms, with dilatation, pneumonia, kidney and other complications. A better feeling of security will exist when the cause of the toxemia is generally known—not only because the toxic condition may be corrected, but also because preventive measures may be utilized.

Acute intoxication of the alimentary tract has been thought to be some form of infection, but scientific workers in this field have been unable to agree on the group of micro-organisms responsible for the toxemia.

The ease with which tissue breakdown occurs in the alimentary tract, with resulting toxemia, led to more direct and definite methods of investigation. The results have shed a flood of light upon these serious symptoms, especially the alarming symptoms associated with abdominal operations.

Similar symptoms and conditions arise where no operations have been attempted, or even contemplated. It was observed that the epithelial tissue of the alimentary tract undergoes disintegration from comparatively slight injury, as occurs in gastric dilatation.

Removal of this disintegrating material from the fasting stomach, by the writer, was found to be highly toxic on injection into animals.[34 43 74] I also found, (1903), that the injection of the extract from the mucous membrane in five experiments caused a destruction of gland tissue of the stomach. The necrosis was especially noted at the lesser curvature and in the pyloric region of the stomach.[71] Injection of the dried homologous mucosa in suspension with non-specific colloids added to low concentration of the mucosa extract was also found highly toxic.[106 104] The intraperitoneal injection of gastric muscle extract caused general symptoms with vomiting and atonic dilatation. Laprotomy 27 hours after the injection showed gastric retention with dilatation.[71]

In all my experiments with tissue extract, either extracted with water or burned to an ash and injected or by absorption from tissue autolysis, gastro-intestinal stasis was produced. This varied in degree from simple atony to complete acute dilatation.[36 60 71 82 108 109] When retention occurred this caused further tissue breakdown and general toxemia.

From the fasting atonic stomach with retention the degenerating gland cells were recovered and examined.[27 29 39] This material passed through a Pasteur filter was found to be very toxic[10] when injected into animals. Degenerating cells of the gastric wall were observed by histological examination.[70 109] It was shown that the tissue toxins of cell degeneration pass into the circulation and cause general toxemia.[58 73 77] These toxic products may also be found in the stomach and are the result of the autolysis of the gastric wall. They are highly toxic when injected into animals. This may partly account for the value of gastric lavage for gastric symptoms following surgical operations.

As to the cause of the dreaded general condition with gaseous distention many different opinions have been expressed. These beliefs are chiefly founded on clinical impressions which are empirical or on personal opinion without any evidence. This form of toxemia has been subjected to careful experimental study. The following was one of the earlier observations of the writer.[34 74 109]

The toxic effects of filtered and sterile stomach contents from the fasting stomach of pathological gastric cases was considered by the writer in 1896 to be due to albuminoses or other proteins.[34 75]

The separation and study of the toxins from the stomach by "fractional coagulation" was the writer's method adopted by Gillespie[15] of Edinburgh (1901).

Local effects of mustard oil, chromic acid, nitrate of silver and tannic acid in the stomach caused effects similar to burns in producing toxemia.[29 33 68]

As noted clinically in burns, and also by experiment, when the tissue is charred to an ash it is highly toxic, and toxicity of this ash excludes the foreign protein theory of tissue toxemia.

The release of the salts of the cells which resist fire and carry the toxic moiety is all that is required to initiate normal and pathological reactions. It is this principle that must be kept in mind in all animal experiments and the clinical application of this active substance we call "cytost". Surgeons are constantly contending with sudden lung congestion, liver, alimentary tract and kidney congestion. These organs, except the tissue framework, are originally derived from the cells of the endoderm, which is extremely sensitive to homologous "tissue toxin".

The Cause of Splanchnic Reaction. Clinical experience and experimental research harmonize in this specific and fundamental reaction, namely, the immediate response of splanchnic congestion to tissue toxin. This occurs as soon as the released tissue extract reaches the splanchnic zone. This was particularly emphasized in all my early researches[34 36 39 42 43]. It is a daily occurrence in all surgical work where the surgeon may view this "living pathology". The direct relationship of tissue toxin and splanchnic stasis is of intensely practical as well as scientific interest. This reaction is so universal in every stage of growth that we must go back to the embryonal stage for its solution.

In the toxemia of pregnancy our experiments have revealed that of the three embryonal layers the endodermal cells or splanchnic area are first involved *pari passu* with the mother.

This is made more clear in a recent publication by the writer[120] based on some early experiments and recent researches on "The pathological reaction of tissue extract (cytost) liberated in pregnancy, toxemia of pregnancy and shock."

The Reaction on the Endoderm. The tissues from the three embryonal layers, ectoderm, mesoderm and endoderm react differently to the "tissue toxin" or cytost. When the toxin was injected into the muscle or mesodermic tissue a local reaction took place (see protocol), but the systemic or general reaction does not occur until the toxin reaches the cells derived from the *endodermal* layers of the embroyo. The organs that are the offshoot from foregut, the

lungs; midgut, the liver; and hindgut, the kidneys; designated as the splanchnic or visceral area or splanchnic zone, show the maximum reaction.

The dried residue of serum and cell substances was made into a 2% suspension with .8% salt solution. Dilutions of 1/20, 1/50, 1/100 and 1/200 were then used. One cc. of the above mixtures was injected intravenously into young rabbits. When anaphylaxis occurred within three minutes this was regarded as a positive reaction. "Slight convulsions and recovery, or death delayed from 24 hours to one week, were considered for this quantitative work, negative. It was conclusively shown that dilution of tissue toxin up to 1/200 produced shock and death (1914)[106] (1917)[110] as shown in the following table indicating that the tissue toxin derived from the mucous membrane of the upper alimentary tract is specific.

Titre of injections of homologous tissue extract from mucosa and submucosa, compared with venous blood:

Dilutions							
1/20		+		+		+	—
1/50	Muc and	+	Submucosa	+	Mesenteric	+	Femoral —
1/100	Submuc	+	Zona Trans	+	Veins	—	Veins —
1/200		+	Formas	+		—	—

But the question arose, why does the tissue toxin produce such a profound effect upon the cells of the splanchnic area with such marked agglutination of these cells and fluids? It seemed to be elective. The sensitive splanchnic zone received the brunt of the tissue toxin and it was explained as follows:

"In the study of inflammation of the stomach in the embryo, it seems that the endoderm is far more sensitive to irritation than the ectoderm" (1896).[39] "The action of this high temperature directly upon the vessels within the viscera is prompt." "The effect by direct methods of treatment upon the endoderm or inner cylinder of the body is more decided. Students of embryology as well as clinicians will recognize this" (1897)[42*]

The elective or specific effect of cytost was demonstrated in all our subsequent experiments. This fact of the sensitive endoderm has lead Pearl to conclude that "the figures make it clear that man's greatest enemy is his own endoderm."[19]

General Pathology Affecting the Splanchnic Area. The kidney lesions, which are the renal expression of the splanchnic reaction, result from some profound physical effect on endodermal cells which is transferred to the tissue fluid and blood. By careful experiments in vivo and in vitro, it was demonstrated uniformly that this was a phenomenon due to tissue toxins and caused agglutinations or adsorption of the cellular protoplasmic substances, and of the tissue fluid and blood as shown in 1903[73]. "Agglutinins, precipitins and hemolysins were demonstrated by the effects of the tissue toxins. Blood coagulation was prompt. The blood corpuscles became thereby contiguous in the capillaries. All this accounted for the splanchnic stasis". (See protocols Nos. 3, 4, 14, 17, 21. "Shock produced by General Anesthesia," *Journal of the A. M. A.*, May 2, 1903.)

The above protocols show the effect of heat applied within the splanchnic area and it is compared with the effect of visceral exposures during abdominal operations.

Clarity of vision sufficient to correct two ideas will make it plain that the experiments with heat application within the stomach and colon, also to the exterior coat of the peritoneum, may be compared with the effect of desiccation of the viscera by air. In other experiments I found the gastric contents and the peritoneal fluid after injury to be highly toxic on injection into normal animals[34 43 69].

Microscopic examination of injured tissue showed that autolysis had occurred[71]. The cells were observed expelling their contents, due to the increased permeability of the cells. This may be caused by heat application or exposure of the delicate cells to the air. By keeping this fact in mind we may pass to the next step in which other agents, like burns, produce the same effect.

Burns and scalds, as now generally admitted, produce disease and death by releasing the specific poison from the tissue. Chemical cautery, sulphuric acid, for example, chars the tissue the same as the actual cautery. Morton[18] showed that the action of corrosive acids and caustic alkalies upon the tissue is similar to that occasioned by the application of heat. The local destruction of chemical cautery may destroy every vestige of organic matter of the tissues and what remains is the inorganic salts which carries the toxic moiety. (See protocols).

The effect of corrosive and caustic agents varied with the degree of cell injury from the escape or extrusion of the cell contents to the complete charring of the tissue cells.

Experiments on the Gastric Mucosa.—Applications of the agent to a definite locality on the gastric mucosa was made with a revolving "sound" or applicator called the gyromele. This instrument was devised by the writer in 1893.[26 29] The tube and cable were inserted into the stomach, the sponge was exposed and gentle revolution of the instrument indicated by external palpation in its exact locality within the stomach or duodenum. After applications of the various agents were made the exfoliated cells of the mucosa were easily recovered for examination. Deep tissue changes were examined microscopically by removal of small bits of the gastric mucosa with "nippers" devised for this purpose.[26 29 31 32 39] Local and constitutional reactions were observed in both the acute and chronic stages. The agents used were corrosive acids, caustic salts, as nitrate of silver, tannic acid, mustard oil, oil of cloves, chloroform, ether, alcohol, etc. These agents produced local changes as follows: first, severe action caused cell necrosis; second, milder agents caused escape of cell contents. The local reaction varied with the degree of cellular substance which was liberated in the mucosa by the agents used. Thus every grade and degree of reaction, acute or chronic, could be produced at will.

These direct observations of the cells showing increased permeability produced by a number of agents gives us a vision of the beginnings of all biological reaction. This is unique in the constitution of living cells. Its very uniqueness constitutes its attraction for we are impelled to take the next step, which is observation of the effect of the released cell contents upon adjacent cells. We may here see the whole in one vision instead of separate parts.

It was seen in the stomach cells that as soon as their contents were set free by the agents used the released cell contents affected at once the living cells. In high concentration of cell contents it caused the death of cell and various grades of cell necrosis was seen to follow. Metamorphosis was observed in which pepsin cells were transformed into mucin-forming cells. In other areas where the concentration of tissue or cellular extract was low the effect

* A practical suggestion may be noted here. The failure to obtain the full benefits of heat application to somatic area need not cause surprise. The writer demonstrated many years ago that heat must be applied within the abdomen to directly act on the splanchnic area (see literature and methods).[36 54 69]

was not retrograde, but caused reconstruction mitosis, with rapid cell division [26 1:1 82 89].

On removing a section of the gastric wall remote from a disintegrating tumor of the stomach the gastric cells were found to be in a state of rapid proliferation because, as then stated, the products of the disintegrating cells stimulated mitosis. The cells adjacent to the tumor showed necrosis due to the higher concentration of the cellular contents of the disintegrating tumor cells acting on normal cells[30]. Now these observations in various forms have become the center of interest in modern biology.

It requires no further presentation of facts to indicate to the surgeon's mind that he is dealing with the phenomena of the cells, not of the body as a whole, nor of a single organ as the kidney, liver or lungs. The latter are simply the aggregation of cells. (See protocols at end of paper.)

Tissue Extract Named Cytost.

In a series of biological experiments on cell division, including the phenomena of wound healing, I have shown[120] that only the substance that may be reduced to ash is the normal stimulant of cell growth. Only the salts of tissue cells is required to excite cell division and metabolism, but when higher concentrations were used it caused death of the cells. This "cell extract" under different names, according to the form of the writer's experiments, is now named "cytost" (*cytos,* a cell) and the opposing antibody "anticytost." The summary and conclusion of the writer[120] was that we have shown by experimental research covering a period of twenty-five years a new biological principle: that cell extract, which we have named "cytost", excites cell division and metabolism in biological concentration and in high concentration produces disease and death.

Low concentration of cytost is physiological; medium concentration of cytost is pathological; high concentration is lethal[120].

Autolysis.

So far the consideration has been that of various agents, mechanical, physical and chemical, which release cell contents. Cytost thus produces the biological reactions and the abnormal conditions with kidney lesions. These cell injuries may be considered as conditions that are *produced,* while autolysis is a condition that may be *induced,* as in partial or total starvation. Withholding all food results in tissue breakdown (autolysis) to supply nourishment. Partial starvation either in quantity or in quality also causes tissue autolysis to supply the caloric defect of "vitamine" deficit.

When autolysis occurs it releases cell contents or cytost. This is what is known as toxemia of starvation. The kidney lesions by induced autolysis from total or partial starvation were demonstrated in my research on peptic ulcer[81 88 92]. They were shown to be due to general autolysis induced by adding extracts to the regular rations. This caused interruptions of digestion, absorption and assimilation, hence autolysis follows to supply the nutritive deficit of the body cells..

The German surgeons complain that the starvation of their people has enormously increased the cases of peptic ulcers. Kidney lesions were a part of this general "constitutional disease".[124 125]

Local cellular starvation always occurs in the tissue in which the circulation is interrupted, or cut off, as in wounds, local starvation resulting in autolysis and the release of the cell contents, cytost. This may result in shock and kidney lesions. Bosquette[17] found, as surgeons have reported from the battlefield, if the tourniquet was left too long on the limb it resulted in shock and death. It requires now no further discussion to explain the reason. The starvation and asphyxia of cells locally always results in autolysis and release of cytost from the cells.

Autolysis of Pregnancy. Many kidney lesions with low resistance are traced to congenital disorder and are classed as "surgical risks". Individuals who show low resistance traced to congenital origin are now known to be the result of the toxemia of pregnancy. Virchow was the first to trace this relationship, even though the kidney lesion appeared in the adult period.

Effect of Cytost on the Egg and the Growing Embryo. A large variety of animals was used in these experiments. As many biologists have noted, the specific effect of tissue extract in the egg excited reaction, so, in addition, I studied similar effects on the egg and embryo produced in the pregnant animal, and by inducing autolysis by various methods of partial starvation, caused tissue breakdown and the toxemia of pregnancy. General symptoms were of the usual character observed in all toxemias of pregnancy with grave kidney lesions in both mother and embryo. Colonies of several thousands of mice that had been under observation for seven years were studied. Thus the death rate was established. The record of morbidity and mortality was high. By injecting homologous (mouse tissue extract) cytost of high concentration into pregnant animals a very high death rate resulted,—95% death rate of both mother and offspring. Pathological findings were general congestion and kidney lesions. In another series the male and female were immunized by active and passive methods. This prevented the disastrous pathological results.

In the following table is presented a further report of one investigation with this mouse colony. Both male and female were injected with low concentration of cytost to induce immunity. When these females became pregnant the injections of low concentration of mouse cytost was continued, ten days to two weeks apart. This induced active immunity. In another group of pregnant mice passive immunity was produced by injecting anti-cytost.

Virchow explained that the toxemia of pregnancy with kidney lesions, (1858)[34] was due directly to toxins in the blood acting on the renal cells. These toxins in the blood were attributed to substances that were formed or liberated by the tissue change of destructive metabolism, which is increased during pregnancy and which is commonly expressed as tissue breakdown. In 1857 he said that "A cell was dead when it ceased to function as a cell", and that it then became "altered albumin."

Ribbert showed that "if primarily the local tissues had undergone injury, there is a vital manifestation on the part of the whole body".

If Virchow and Ribbert had only gone one step farther and simply injected the "altered albumin" of the necrosed cells into animals, they would have unfolded much of the mystery that lies at the foundation of all biological and pathological reactions.

By observing the results of injection of merely the watery extract of tissue cells, these great pathologists would have struck the mountain peak of biology and traced it down into the valley of pathology and

death. It was reserved for modern research to develop the relation of biologoical reaction to tissue extract now named "cytost."

A recent presentation by the writer on "The pathological reaction of tissue extract (cytost) liberated in pregnancy"[123] showed that the effects of cytost were produced by injecting rabbits with mouse tissue extract or cytost. The rabbit's blood contained the anticytost for mice. The result was that none of the mothers or offspring died except one which was eaten—cause unknown. The young went into the second generation without death. We find no deaths until the third generation is reached. The mortality rate was, however, very low. Space does not permit for a more detailed report.

The following condensed table gives number of mothers, births, dates, etc.:

PROTOCOLS.

Mice—7 females, 2 males, receiving antigen—2 drops of 1-10 dilution injected subcutaneously every 10 days.

Date of Injection	Amount	No. Mice Injected	Births**	Deaths
12-9-22	2 drops	9	12-5-22 3	
1-11-23	2 drops	9	1-16-23 4	
1-19-23	2 drops	8*	1-18-23 7	*1 mouse eaten by others
1-29-23	2 drops	8	1-25-23 5	
2- 9-23	2 drops	8	2- 2-23 6	
			2-13-23 1	
			26	0

**One newborn mouse died—cause unknown.

Mice—8 females, 2 males, receiving rabbit serum—1 drop injected subcutaneously once a week.

Date of Injection	Amount	No. Mice Injected	Births	Deaths
12- 6-23	1 drop	10	12- 4-22 7	
12-13-23	1 drop	10	12-24-22 8	
1-11-23	1 drop	10	1-28-23 7	(1 newborn mouse)
1-19-23	1 drop	10		
1-29-23	1 drop	10		
2- 5-23	1 drop	10		
2-14-23	1 drop	9		
			22	1

*1 mouse not injected because of raised spot at site of previous injection.

Report for December to date, of deaths of offspring of 10 mice that had been immunized. Immunity does not subside until the third generation.

Deaths	Generation	Autopsy
(1/10/23)—1	3rd	Liver enlarged — intestine mucoid. Heart and lungs normal.
(1/15/23)—1	3rd	Stomach hard, other organs normal.
(1/16/23)—1	3rd	Lungs greatly consolidated. Other organs normal.
(1/19/23)—1	3rd	Lungs consolidated. Other organs normal.
(1/20/23)—1	3rd	Destroyed—not autopsied.

Comment. These additional protocols taken together with those already published[123] give most valuable facts on transmitted specific immunity from mother to young. As these protocols show, when both the males and the females are immunized the death rate falls from about 30% to near zero. The immunity apparently continues through to the third generation. The morbidity and mortality is even then reduced much below that of its controls. By immunizing the offspring a still higher degree of immunity is established.

Hornell and Erby[20] found that the serum of offspring contained immune bodies from the mother, but less than the mother.

A summary of experiments on the toxemia of pregnancy was made in the paper published this year,[123] read at the New York Academy of Medicine before the Section of Gynecology and Obstetrics. In the discussion Prof. Pike called attention to the principle of tissue toxemia and immunity, the biology of which has been fully demonstrated in the war. He concluded: "All this time we had growing up here in America work leading exactly to the same conclusions. Because of this peculiar blindness we did not see it. Now I am engaged in translating Dr. Turck's results into my own language so that I can understand them". It has been the attempt of the writer to help the surgeons also to see and clothe the language so that the great principles of cytost—anti-cytost reaction would appear familiar to the practicing surgeon as well as to the biologist.

This brings us to the practical application of this principle. The protocols, with the references to the biological work, have been placed at the end of this paper for those interested in biology applied to surgery.

Summary and Practical Suggestions in Treatment.

The biological balance that sustains the organism against cytost intoxication is anticytost which we have recovered from the blood in animals and in man. This was further placed on a sound and practical basis by investigating the effects of cytost liberated in mothers on offspring. This was compared with the effect of increased anticytost, the mother producing immunity against cytost, thus protecting both mother and offspring. The recent publication by the writer on the pathological reaction of tissue extract (cytost) liberated in pregnancy[123], showed the effects of cytost on the egg and growing embryo, —also the comparison with the effect in mothers actively immunized by cytost injections and passively immunized by anticytost.

This work explained more clearly the practical benefits which I have reported in the use of methods of producing biological immunity in animals, including man. This may be used now to explain scientifically, what has been attempted empirically in both active and passive immunity. The actual cautery in surgical practice and its modification for general stimulation is too well known. Burns produced by diathermy, *x*-ray and radium, are the more modern application of this ancient method of treatment.

Active Immunity.

Active immunity has been accomplished by either injecting tissue extract into the body in low concentration, or by liberating the tissue extract (cytost) by some form of cauterizing agent. As we have seen, severe burns produce shock and death, but slight burns do not. The effect of slight burns was to stimulate the living cells. Cauterization was first used by the Chinese by puncture with hot needles, nearly 3,000 B. C., or by applications of a burning wick dipped in oil. The Ku treatment is the Japanese adaptation of this. The Germans took up the Chinese method in the 18th century and used it in veterinary practice, known as "firing the horse's hoof". Acupuncture by the Germans was a brutal development of this form of tissue injury. The effect of all these methods are practically the same—to release the tissue extract from the tissues. The exposure of the peritoneum to the air, as the writer has shown, causes desiccation of the tissue similar to a burn. This has been used for the surgical treatment of tuberculous peritonitis.

Burns produced by diathermy, radium and *x*-ray are used now more exactly than formerly, with less danger, and more effectively, since the scientific

facts learned from experimental research are absorbed by the practitioner. All these methods combined were recently reported in Proceedings of the French Congress at Strasbourg, 1921,[124] and Paris, 1922[125].

Injecting a fraction of a cubic centimeter of chloroform into the tissue causes liberation of cytost, and is used in active immunity. Now that we can use the human tissue and extract cytost in aseptic ash, the treatment, as an exact measure, is placed on a scientific basis.

Passive immunity by injecting human anticytost, is the practical result of this scientific research. Horses are injected with human tissue extract cytost for one year. The blood contains the human anticytost and the ash of this horse serum contains the same human anticytost. By combining both active and passive immunizing methods, a high degree of immunity is established with a wide range of application.

Experiments.

The doubtful and difficult questions which every surgeon must meet are in the healing of wounds, of transplantation of tissues, the effect of anesthetics and of the surgical operation and lastly the many obscure surgical diseases in which kidney lesions indicate general reaction. We must turn for aid to the same recourse that is had by all biologists in the study of cytology. We have looked into the cells and have seen the reactions there that are normal and those that are abnormal. The biology of surgery then becomes of more value to the surgeon than its mere mechanics or technic of operation. To know this better we may compare our observation on animals and man with studies on the behavior of the single cell of the organism. Let us see what modern biologists have taught us concerning the single cell, so we can see the whole in one vision.

It is clear now that the aim of investigators has been to determine the specific agent that starts cell division and metabolism for it must be at the very foundation of biology upon which medicine and surgery rest. The entire life process, normal and abnormal, is involved in cell division, growth and metabolism.

Conklin[24] used mechanical, physical and chemical agents on the egg cell of crepidula. He saw that it made no difference which agents he selected so it released the "cell sap" as he called it, within the cell. Then the cell began to divide and metabolize. He caused all forms of normal and abnormal changes by varying the degree of the "flow of cell sap". Osterhaut[22] simply touched the large cell, *Greffethsia*, at one end, observed under the microscope. At once the red pigment began to diffuse out into the surrounding protoplasm. Osterhaut says "At the point where the cell is touched, pigment, and probably *other substances* are set free, diffuse out and set up secondary changes as they progress. Lille found the permeability of the cell was increased and that the cell contents act as a catalyzer and caused the cell reaction. Loeb[25] had reproduced the phenomena with accurate measurements qualitatively and quantitatively.

Childs[23] found that the water in which large stocks of *planaria* had been kept and which therefore contained the soluble products of metabolism, produced essentially the same results as cyanide, ethyl alcohol, ether, chloroform, chloretone, acetone, and other narcotics. The soluble metabolic products produced senescence and rejuvenesence in the *planaria* the same as the reagents mentioned, varying in reaction according to the concentration used.

It was found by the writer that chloroform and ether caused cell necrosis and escape of the soluble products of the cell into the tissue. This caused shock and death. The cell toxin was recovered in the blood and injected into animals. This also caused shock and death.[73] (1922) Mary Grace Springer,[21] studied the effect of embryological extracts of *arabacia* upon developing eggs of the same species. High concentration resulted in "cytolysis and complete arrest of development, or the production of abnormalities, similar in certain respects to those produced by acetone or other chemical reagents."

Protocols.

The following protocol shows the specific effect of "tissue extract" which is extracted by incineration to an ash method. The ash of cat tissue in a solution, 10 parts of water to one of ash, was used and compared with the effect of lion tissue of the same concentration. Injected 1/2 cc. of cat tissue ash (cytost) (10/1) into the thigh muscle of cat and 1/2 cc. of same concentration of lion cytost into opposite thigh muscle. This was allowed to remain three hours. The muscle area injected was resected and sections made for microscopical examination. Cell mitosis was very active in the tissue of the leg injected with cat tissue ash. The muscle tissue injected with lion tissue ash showed no specific reaction. The difference of the action of cat tissue ash in exciting the cell division was in contrast with the negative effect of an alien animal's tissue. This indicated tissue extract (cytost) is specific to the species. The lion, though of the feline group shows its alien character. This conforms with our other biological experiments in which animal tissue was shown to be specific to the species.

The conclusion is inevitable that all foreign agents, physical or chemical (including foreign tissue extract) must first release the specific cell extract within the animal to produce the biological reaction and the production of abnormalities.

My experiments on wound healing[120] proved that active and passive immunizing methods with cytost and anti-cytost injections hastened the rate, shortened the time and improved the character of healing.

The clinical application of this method by Kellogg confirmed the experimental findings in his hospital report[119] [119] [115] (Kellogg—Rate of cell necrosis, etc., *N. Y. Medical Record*, March 22, 1919. Kellogg—Treatment of Borderline Cases, *N. Y. State Journal of Medicine*, March, 1920.)

The following résumé of the experiments, while condensed, completes the evidence on these principles. The methods as outlined, give the essential findings and results.

RÉSUMÉ OF CYTOST-ANTICYTOST PHENOMENA.

A. Agents used to produce Homologous Cytost.

1 Burns
2 Chemical { Alcohol, Ether, Chloroform, Oil of Mustard
3 Mechanical { Trauma, Surgical interference
4 Electrical
5 *X*-ray
6 Exposure to elements (pressure)
7 Ferments (mixtures)

B. Modes of Administration.

1 Subcutaneously
2 Intramuscularly
3 Intravenously
4 Intratracheal
5 Intraspinally
6 Intraperitoneally
7 Spraying of Paws (Pneumonia Exp.)
8 Intraarticular
9 Intraosseous

Note: In the ensuing tabular résumé one or more of the agents were used and different modes of administration were used for each agent.

C. Homologous Cytost (for controls).

8 Cat cytost
- a Ash sol. 10/1 112 to 1 cc.
- b Muscle ext. 10. 1 cmc.
- c Berkfeld filtrate of b.
- d Alcoholic ext. placenta.
- e Alcoholic ext. fetal tissue.

D. Heterogenous Cytost.

9 Horse tissue cytost—prepare same as b.
10 Beef
11 Lion
12 Dog
13 Guinea-pig.

Cytost is a specific agent derived from broken down tissue (isoautolized). It is inorganic in constitution (because it is obtained from tissues fired at 300° C.) and it causes reaction in the homologous body.

Anti-cytost is obtained by immunizing animals against cytost. It is the antipod, a counter steriosis or a counter electrical charge which neutralizes cytost in vivo and in vitro.

Condition Produced		*No. of Cases*	*Clinical Symptoms*	*Post-mortem Pathology.*
1 a Nausea of pregnancy		9	Vomiting Convulsions Anorexia Eclampsia	Glomerulitis Acute nephritis Subacute nephritis
b Kidney and pregnancy		23	Kidney retention Shock Abortion	Chronic nephritis
Controls		12	Retching Slight vomiting Negative nephritis	Negative or slight nephritis (when destroyed to determine pathology.)
2 Pneumonia	Acute	43	Cough Congestion	Congestion Acute pneumonitis
	Chronic	77	Râles Dullness, etc.	Lobar pneumonia Lobular pneumonia
	Controls	100	Anorexia Vomiting	Broncho-pneumonia
	This figure is small and only an estimate.		High temperature Dyspepsia, etc. Death in ½ minute to many weeks.	
3 Shock		44	Fall in temperature Unconsciousness Rapid pulse (weak)	Cardiac dilatation Stomach dilatation Congestion of gastro-intestinal tract, liver, kidney and lungs and adrenal gland
	Controls	25	Dilatation of stomach Cyanosis Death usually in ½ minute or less in acute form	
4 Arthritis		10	Periarthritis Lameness	Thickening of synovial membrane and ligaments
	Controls	3	Swelling Ankylosis Anorexia Synovitis	Periarticular effusion Atrophy of muscles Anemia Inflammation of hyaline cartilage and its erosions Congestion of epiphysis Osteomyelitis
5 Anaphylaxis		16	Anorexia Urticaria High temperature Chills	Pneumonitis Gastro-enteritis Acute nephritis Cardiac dilatation
	Controls	8	Vomiting Shock Death	Dilatation of stomach Shock
6 Gastric Ulcers		16	Vomiting Pain Anorexia Emacution Constipation	Gastric ulcers Duodenal ulcers Erosions
7 Arteriosclerosis				Arterial fibrosis Cardiac hypertrophy
(Metabolism)		75	Cardiac hypertrophy Pulse hard, rapid High blood pressure	Chronic nephritis Atrophy of the liver Bone changes
	True arteriosclerosis	6	Pain Animals unable to jump Senescence	Pericarditis Pneumonitis Albuminuric retinitis
8 Paralysis and chorea		24	Delirium Shock	Peripheral, Spinal, Cerebral } Degeneration, Congestion
	Controls	10	Coma Urinary incontinence Paralysis and its associated characteristics Muscular atrophy	Muscular atrophy Paralysis of internal organs

Note: This table represents the most important part of the recent work done at the writer's laboratory and does not include the more thorough technic, pathology, detailed classification of symptoms, etc. This tends to give the reader a rapid outlook on the scope of this work and does not pretend to give a quantitative résumé, as this would make a table too lengthy for the purpose that is intended.

CONCLUSION.

1. "General constitutional" reaction must first occur to produce permanent kidney lesions.

2. The general reaction is caused by tissue extract named cytost.

3. The biological and pathological effect is directly and specifically on the endoderm or splanchnic area.

4. Active and passive immunity is the natural resistance produced by cytost and anticytost.

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LAMINECTOMY IN THE PARAPLEGIAS OF POTT'S DISEASE.

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Tuberculosis of the spinal column (Pott's disease) lies chiefly in the domain of the physician and the orthopedic surgeon. Not infrequently, however, through involvement of the nerve structures, it becomes a matter of concern to the neurologist.

With modern methods of treatment the orthopedic surgeon can usually confine the disease process to the vertebrae in which it originated, and effect a cure without the cord or its roots becoming involved. But if the disease is rapid in development, with much bone destruction and a large accumulation of carious material, the sudden collapse of the affected vertebrae, or compression by caseous matter, may acutely affect the cord and cause marked and even widespread paralysis below the level of the lesion. Brainerd Whitbeck, who has had an unusually large experience in the treatment of spinal tuberculosis, estimates that 5-8% of cases are complicated by some degree of nerve involvement.

In many of these conditions the paralytic symptoms gradually disappear with the usual treatment of rest in bed and hyperextension of the spine. But there can be no doubt that too often this treatment alone is persisted in for too long a time, so that with a continuous and often an increasing compression, secondary degeneration occurs in the nerve structures and consequent irreparable damage.

There is an element of doubt in every case of spinal caries with cord involvement, after several months of treatment by recumbency and hyperextension with persistence of the paralysis, as to whether the paralysis requires laminectomy or will recover if the conservative treatment is continued. For the cord involvement may be due to bone compression, abscess formation, pachymeningitis, edema, or bloodvessel obliteration with secondary cord softening.

The majority of these conditions are best treated conservatively. Others require laminectomy, and some will show little improvement in the paralysis with any form of treatment. These are the cases with secondary degeneration of the cord fibers. It is my conviction that many cases of the latter type could be removed from the incurable class by earlier laminectomy. This does not mean indiscriminate operation. As said above, the majority of these conditions are best treated conservatively, but where operation is indicated it should be performed more promptly than it is at present.

The difficulties confronting the surgeon concerning the necessity of operation are enhanced by the fact that if he does decide to operate, he is entering an area the disease process in which, even though latent, or perhaps "cured," may be relighted by his invasion. But the chance of this occurring is slight and should not deter one if the indications for operative relief are imperative. Integrity of the nerve structure is more important than that of the bone.

Caries may attack any part of the spinal column, but is found more often in the thoracic and upper lumbar regions. It is in the thoracic region that nerve complications more often occur. Spinal tuberculosis is largely a disease of childhood, occurring most frequently from the second to the tenth year. In adults it may or may not be secondary to tuberculosis elsewhere in the body, and the course of the disease is more prolonged than in children. It attacks chiefly one vertebral body or one joint and the disease may not extend beyond this: but as a rule from two to four vertebrae are finally involved. If untreated its location is marked by the appearance of a knuckle on the spine, due to the breaking down and collapse of the affected vertebrae. Rarely it appears in the transverse processes or arches and its site may be indicated by a depression due to the absorption of the laminae and the spinous process.

When situated in the upper cervical vertebrae, the first signs are pain in the back of the neck and head (occipital neuralgia). The head and neck are held rigid and in turning the patient revolves the body as a unit. He avoids jarring movements, and at rest often supports the head with the hands. If

the cord or the roots become involved the signs present depend on the degree of compression. Intractable neuralgia, hypesthesia and even anesthesia occur in the distribution of the affected roots, and there is usually tenderness on pressure over the spinous process of the affected vertebra and on pressure over the transverse processes. On the motor side disturbances of deglutition and respiration are frequent, and if the compression is severe there is weakness and spasticity of the arms and legs, but the paralysis in the extremities is not as severe as that seen in caries lower down. The above relates to an extreme type of cervical caries which fortunately is not often seen. Tuberculosis occurs less often in the cervical than it does in the thoracic and lumbar regions. It is usually recognized early, and because of the small amount of weight the spine in this region has to bear, there is little deformity and few abscess formations; and, too, it heals more quickly than spinal caries elsewhere.

If the disease is situated in the mid-cervical or lower cervical vertebrae there will be pain and rigidity and a tendency to carry the head in a fixed position. If the nerve roots or the cord become involved, there will be atrophic flaccid paralysis of the arms with spastic paralysis of the body below the site of the disease. Motor power is always more impaired than sensation, but areas of anesthesia or hypesthesia are usually present. As the cord is rarely completely interrupted, the atrophic paralysis of the arms is usually confined to several muscle groups. Even in conditions of only partial paralysis of the arms, the sensitiveness of the cord motor tracts to pressure is such that extreme spasticity and paralysis of the legs may be present. In severe compression of the cord, disturbances of bladder and rectal control are found.

In caries of the upper thoracic vertebrae the first sign is pain in the distribution of one or both of a pair of intercostal nerves, often severe cramping pain in the lower chest and epigastrium that leads to diagnosis of indigestion. Caries in the lower thoracic vertebrae causes girdle pains or the disappearance of the abdominal reflexes. As compression increases we find increasing motor and sensory impairment, paraplegia with exaggerated reflexes and spasticity, hypesthesia and even anesthesia, impairment and even loss of bladder and rectal control.

When the disease attacks the vertebrae opposite the lumbar enlargement of the cord, if the nerve structure becomes involved, there is present not spastic but atrophic, flaccid paralysis, sometimes complete but often confined to various muscle groups. This is nearly always accompanied by disturbances of the sphincters whose spinal centers lie in the conus.

In caries of the lumbar vertebrae surrounding the roots of the cauda equina, the nerve involvement is confined to one or both of the pair of roots emerging near the affected vertebra. Their involvement is characterized by pain, hypesthesia and atrophic motor impairment in their area of distribution. Unless the bone destruction is extensive with the accumulation of much caseous material, it is rare for more than one or two pair of nerve roots to be involved.

Tuberculosis of the sacrum is situated usually in the sacro-iliac joint, and is characterized by swelling, tenderness and pain in the joint, when pressure is applied to the iliac crests. Caries of the sacro-iliac joint is usually confined to one side and is accompanied by some of the following nerve complications —pain and sensory impairment in the perineum and surrounding areas, impairment of sphincteric control, irritation and sometimes paralysis of the sciatic nerve with disappearance of the ankle jerk, hypesthesia, and at times flaccid paralysis in the muscles supplied by one of the subdivisions of the sciatic nerve.

Diagnosis: As a rule the diagnosis of spinal caries with involvement of the nerve structures is not a matter of great difficulty. It has usually been known well in advance of the appearance of cord symptoms that the patient has Pott's disease, and he probably is under treatment at the time of their appearance. Even in untreated cases, and in those in which the cord symptoms rapidly follow the onset of the bone tuberculosis, the patient's history, perhaps the presence of tuberculosis elsewhere in the body, the spinal deformity or, if this is absent, tenderness of a certain vertebra, and above all, employment of the *x*-rays, combine to present a picture that is usually readily recognizable.

If the disease begins insidiously and involves the nerve structures early, spinal caries may be confused with cord tumor. The absence of bone deformity in tumor and the use of the *x*-rays usually settles the matter. Sarcoma of the bony arches, especially if there is much bone erosion, may present an *x*-ray picture that much resembles that of tuberculosis. As a rule, the pain in sarcoma is much more severe, and the spinal deformity is absent or develops much later, than in caries. The severe pain of sarcoma is not relieved by immobilizing the spine in plaster or on a frame, while such measures abolish pain in caries as a rule,—though Whitbeck says that if abscess is present, the pain may persist for some time after immobilization.

Osteomyelitis following various infectious diseases

or suppuration elsewhere in the body, occurs occasionally in the vertebrae and at times is accompanied by cord involvement. The high temperature, the severe prostration, and the presence of foci in other bones, enables one to distinguish osteomyelitis from vertebral tuberculosis.

Osteo-arthritis of the spine may be confused with caries but the use of the *x*-ray will usually serve to differentiate them. According to Whitbeck, spinal caries, especially in children, is very often diagnosed as rheumatism, muscular or arthritic. The marked muscle spasm in caries and the tenderness of these muscles lead to this error.

MANNER OF NERVE INVOLVEMENT: Nerve root or cord involvement, when it occurs in the course of the disease, is brought about in several ways. Actual compression by bone is not common. The spinal canal is quite roomy, and destruction of bone is usually a gradual affair so that the cord has time to accommodate itself to the moderate angulation that follows. If bone destruction has been widespread and rapid, so that there is a marked angulation, sudden in development, the cord may indeed become severely compressed, but this is unusual. Occasionally a sequestrum pushed out into the spinal canal from a vertebral body may compress the cord. In conditions of bone compression, the signs of cord impairment are gradual in onset and rather slow in development, unless there has been a sudden collapse of more than one vertebrae, in which case the interruption of cord conductivity may be immediate.

Much more common is it to have compression of the cord caused by the progressive accumulation of caseous material in the spinal canal, the so-called "cold abscess". The nerve roots in the vicinity are also compressed and are inflamed and swollen. The carious material resulting from the bone destruction either in the vertebral bodies or in the arches, tends to be thrown into the spinal canal, to lie there until absorption ultimately occurs. Occasionally, however, it breaks out forward and gravitates downward to form a psoas abscess if the lesion is in the dorsal region, a retro-pharyngeal abscess if the upper cervical vertebrae are involved, or a mediastinal abscess if from the thoracic vertebrae. The signs of cord impairment due to the pressure of carious material in the canal are slight at first but progress rapidly and require only a short time for full development of the paraplegia. If compression has not been severe or prolonged and the abscess breaks through the canal, the signs of paralysis will gradually disappear even though the disease process in the vertebrae is unchecked.

Cord impairment may be brought about in still another way. Even in the absence of any considerable amount of carious material, the dura opposite the diseased vertebrae may become inflamed, enormously thickened and with subsequent edema of the cord and impairment of conductivity. The neighboring nerve roots are also inflamed. Or there may be a real pachymeningitis with adhesions and localized collection of fluid (serous meningitis) causing cord compression. The signs of cord impairment thus caused are gradual in onset and slow in progression.

Occasionally, due to meningeal irritation, there will occur in the portion of the cord adjacent to the tuberculous process, a condition of edema of the cord fibers. This may give rise to faint signs of mild cord impairment, such as root pains and slight muscular weakness. This cord edema tends to disappear as the disease process in the bone improves but if it is present over a period of several months, there may result inflammation and secondary degeneration of some cord fibers, presenting a picture of partial transverse myelitis. If the meningeal irritation has been severe, adhesions may form with a localized collection of fluid (circumscribed serous meningitis) which, once established, tends like any encapsulated condition within the dura, to persist in some degree unless relieved by surgical measures.

The cord can be involved in still another way. The disease process may set up an obliterative arteritis in the vessels of the cord, causing secondary degeneration or softening of the nerve fibers, a limited myelomalacia. It is rare for the disease process to directly attack the cord, that is to set up a tuberculous process in the cord itself. The signs of cord impairment due to secondary degeneration or to a myelitis are gradual in onset and slow in development, but do not disappear when the disease process in the bone is cured.

TREATMENT: If in the course of spinal caries signs of nerve involvement appear, the patient should be put to bed with the spine hyperextended on a convex stretcher frame of the Whitman type. The patient should be examined at intervals by a neurologist to determine the exact degree of paralysis present, for these findings will determine in part the future treatment. While it is often difficult to determine the cause of the cord involvement there are several signs that are helpful in arriving at a decision. If there is marked angulation of the spinal column with collapse of two or more vertebrae, and the development of the cord symptoms has been gradual, the condition is probably one of angulation of the cord and compression by bone. It is unusual to have a complete paraplegia as a result of compression by bone.

If the cord signs develop rapidly and the paralysis

is marked or complete, the condition is probably that of cord compression by "abscess",—caseous material in the spinal canal. Abscess formation is by far the most common cause of cord compression.

Signs of cord impairment due to pachymeningitis or obliterative arteritis cannot be distinguished one from the other, or indeed often from those due to compression by bone or by abscess formation, except for the fact that the former do not clear up under treatment. Fortunately these two conditions are of rather infrequent occurrence.

Pachymeningitis is believed by many to be the most frequent cause of the paraplegias occurring in Pott's disease. This belief is based on the result of autopsies, a majority of these disclosing a pachymeningitis and fibrosis of the cord substance. From this it is argued that laminectomy holds but little promise of benefit. But practically all of these autopsies are performed several years after the onset of the paralysis. Very few of these patients die as a direct result of the paralysis. They usually live for several years, finally succumbing to intercurrent maladies. An autopsy several years after cord involvement had occurred, with continued interference with cord function, as for example by bone compression, will naturally disclose a condition of old pachymeningitis and cord fibrosis. The fact that 90% of the paraplegias will recover when treated by recumbency and hyperextension is alone sufficient to offset the belief that pachymeningitis is the chief cause of the paralysis.

Whitbeck estimates that in 90% of patients with cord impairment the paralysis, if treated from its first appearance by recumbency and hyperextension on a proper frame, will wholly or almost wholly disappear. The length of time required for the disappearance of the paralysis depends on the condition causing the compression and upon the institution of prompt and proper treatment. In abscess formation, improvement of the paralysis will begin as soon as the abscess breaks out of the canal or begins to be absorbed. If recumbency and hyperextension have been employed on the first appearance of the paralysis, improvement will begin by the second or third month, and indeed cord recovery may be complete by the fourth month. In conditions of severe compression of the cord, several more months may be required for complete recovery, but there is always some improvement found by the third or fourth month even in the cases of longer duration.

Paralysis due to bone compression even though treated from the onset by recumbency and hyperextension, requires a longer time for disappearance of the nerve impairment, than that due to abscess formation, but the initial signs of improvement will appear about the third or fourth month. Whitbeck has seen paraplegia persist for eighteen months under continuous treatment, but the initial improvement began about the third or fourth month. Even though the bony angulation is extreme, treatment by hyperextension, though it apparently corrects the deformity but slightly, is usually sufficient to remove the compression from the cord.

Naturally, the cord impairment caused by a thickened and constricted dura, or by a pachymeningitis, or by obliterative arteritis will be influenced but slightly by the treatment of recumbency with hyperextension, except in so far as rest with immobilization of the spine will have some effect, together with the normal tendency of impaired structures to recover some function. Fortunately, these conditions form a small percentage of the whole.

As said above, the paralysis should begin to show some improvement, even though slight, by the third or fourth month. This applies to cases that have been treated by recumbency and hyperextension from the first appearance of the paralysis. In patients with paralysis who have been untreated or improperly treated, the time of expected improvement should date from the application of proper treatment, that is, three or four months following recumbency and hyperextension on a convex stretcher frame. If there are no signs of improvement in the paralysis at five months, it is evident that we are dealing with a condition that may require treatment other than hyperextension. In a properly treated case, if there has been no improvement in the paralysis within six to ten months, laminectomy should be considered. It is for this reason that it is important that the patient should be examined by a neurologist at intervals from the onset of the paralysis, so that exact findings of the degree of paralysis can be secured. If improvement is definite but slight at the end of from three to four months, only by neurological examination can it be noted. And the findings at the later months compared with those of earlier examinations will determine whether laminectomy is indicated or whether recovery can be expected with conservative treatment.

Contrary to the belief of some, a laminectomy in Pott's disease is not more formidable or dangerous than in other spinal conditions requiring operation. The disease is nearly always confined to the vertebral bodies and an exploratory or decompressive laminectomy does not disturb or invade this region.

OPERATION. With the removal of the spinous processes and laminae as described in another article,*

* Spinal Decompression—Sharpe. *American Journal of the Medical Sciences*, September, 1919 (Vol. **158**, p. 335).

the cause of the cord impairment will usually be revealed to inspection. It is, in order of frequency, bone compression causing angulation, detritus in the canal, or a thickened and constricted dura. Pulsation is usually absent in the portion of dura compressed and below this point. If there is much carious material or a sequestrum in the spinal canal, this is removed, avoiding invasion of the affected vertebral bodies. If there has been direct compression by bone the removal of the laminae will have allowed the cord ample room.

The dura is usually found enormously thickened and doughy. When it has been the cause of the compression of the cord, there will be found one or more constrictions in this thickened portion. These should be cautiously divided, care being taken to avoid opening the dura. When the disease process is active, and it nearly always is six to eight months after the onset of the paraplegia, the dura should never be opened, and one must be content with what can be done in the spinal canal to remove compression or divide constrictions. Fortunately, in the majority of patients the cause of the cord impairment lies outside the dura.

If pachymeningitis or obliterative arteritis has been the cause of the paralysis, on removing the laminae inspection will reveal no evidence of compression outside of the dura, and as a rule the dura will be seen to be pulsating normally. An exception to this is serous meningitis,—a localized collection of fluid. In the presence of active caries the dura cannot be opened. This can and should be done at a later operation, if the paralysis persists after the disease process has been cured. The laminectomy wound is closed, as described in the previous article. A drain is rarely used, for while the wound heals as readily as in non-tuberculous conditions there is a possibility that drainage might lead to sinus formation. By the use of felt pads on either side of the spine the patient can be returned to the convex stretcher frame to receive hyperextension immediately after operation.

CASE I. A. B., girl, aged 12 years. When 4 years old she fell from a chair striking her back; she did not appear to be hurt, nor in much pain. Four months later it was noticed that in walking or running she held the shoulders in a peculiar fixed position, but there was no apparent deformity. At a hospital to which she was taken a plaster jacket was applied to the upper thorax and worn for three months. Two weeks after removal of the jacket she lost power in the left leg. One week later, the right leg became paralyzed over night. A diagnosis of spinal caries had not been made and for three years the condition was treated as one of infantile paralysis (poliomyelitis). An examination by the *x*-rays revealed Pott's disease, and the child was treated for this the following eighteen months, without any marked improvement in the paralysis. When she was referred to me the paraplegia had been present for seven years.

Status. She was a well nourished girl with a kyphosis involving the first three thoracic vertebrae. There was a complete spastic paraplegia with exaggerated tendon reflexes and double Babinski phenomenon. Extreme hypertonicity of the muscles of the legs and trunk was present, but no contractures, and the muscles were fairly well developed. There was hyperesthesia of both legs and trunk, but no anesthesia even in small areas. The abdominal reflexes were absent. No disturbance of sphincters.

Operation. Laminectomy, January 24, 1919. Through a four-inch incision extending from the seventh cervical spine downward, the laminae of the first three thoracic vertebrae were removed, exposing three inches of the dura. There was no evidence of compression outside the dura and the cord was not markedly angulated. The dura was much thickened over a distance of two inches and was of a peculiar doughy consistency. About the middle of the thickened portion the dura was constricted and at this point there was no pulsation, although both above and below, the dura pulsated normally. The constriction was divided without opening the dura. The wound was closed in the usual manner and a narrow plaster splint applied. Convalescence was uneventful and the child left the hospital three weeks later with no change in the paralysis. Examined two years later, the condition was as before operation, a complete motor paraplegia.

Comment. Although the long duration of the paraplegia (seven years), made it doubtful that operation would accomplish anything, it was hoped that a condition would be found that could be helped. The presence of hyperesthesia rather than sensory loss made this more probable. At the operation it was evident that the cause of the paralysis was within the dura, and I regret now that the dura was not opened. It is doubtful, however, whether this would have disclosed anything remediable and because of the rather inflamed appearance of the dura, it was thought unwise to incise it. Before the operation it was realized that if there was no obstruction found in the canal outside the dura there was little hope of relieving the paralysis, except in the rare chance of finding an encysted collection of fluid subarachnoid. This would have been evidenced by a tense swollen dura, with faint or absent pulsations. The condition was probably one of a partial transverse myelitis, a condition irremediable by operation. But we cannot be certain that this was a myelitis since the dura was not opened, which we can now see should have been done in this case.

Operation cannot promise much in paraplegias that have persisted unimproved eighteen months or

longer under conservative treatment, that is recumbency and hyperextension. And the chances of operative benefit are definitely lessened as the time interval increases. The point to be emphasized is that these patients should not be allowed to drift along down the years without an effort being made to relieve the cord by open operation, after six to ten months of conservative treatment has failed to improve the cord functioning.

CASE II. I. R., female, aged 35 years, a healthy woman, mother of three children. Beginning in December, 1917, she had dull dragging sensations in the muscles of the abdomen, and at times complained of a constriction about the waist. A few weeks later the right leg felt numb and heavy so that she dragged it slightly. On sitting down she had pains in the lumbar region of the back. The loss of power in the right leg was gradual and progressive, the leg became slightly swollen and painful, and five weeks later the left leg became weak, but at no time did she have any pain in the left leg. Shortly after her illness began, she was taken to a hospital where roentgenograms showed partial destruction of the ninth and tenth thoracic vertebral bodies. Spinal tuberculosis was diagnosed, a plaster jacket was applied and the patient was confined to bed for seven weeks. There was a gradually increasing impairment of motor power in both legs during these weeks, and she returned home the latter part of January, 1918. During the following six months the paralysis became worse, so that by August, 1918, there was an almost complete paraplegia.

Examination in August, 1918, showed the right leg almost completely paralyzed from the hip down, the patient being able only to flex and extend the toes. There was anesthesia to touch, pain and heat below the knee, and hypesthesia to above the iliac crest. She could weakly flex the left knee, but all muscular movements of this leg were very weak. No anesthesia on the left side, but hypesthesia to all forms of sensation to the middle of the thigh. The paralysis was spastic in type, the tendon reflexes exaggerated, more so on the right side, and a double Babinski phenomenon was present. The sphincters were normal and the blood Wassermann reaction was negative. Roentgenograms showed bone destruction in the bodies of the seventh, eighth and ninth thoracic vertebrae. There was a knuckle at the seventh and eighth spinous processes.

Operation. Laminectomy, August 28, 1918. Through a six-inch incision the spines and laminae of the sixth to ninth (inclusive) thoracic vertebrae were removed. The bony arches were normal in appearance and apparently were not involved in the disease process. At the point where the knuckle was present there was a marked angular deformity of the spinal canal compressing the cord against the posterior arches. The dura was congested and compressed at this point and pulsated normally only in the upper portion of the wound. With removal of the bone at the point of angulation, the dura rounded out and pulsation returned. A large amount of detritus found in the canal was removed. Because of the activity of the disease process the dura was not opened. The wound was closed in the usual manner and a posterior plaster splint applied. The wound healed by first intention.

Post-operative. Two days after operation there was some return of sensation in the legs. Improvement was gradual and steady so that four months after the operation the patient walked normally and there was complete return of motor and sensory power. Examination three and a half years after the operation showed some rigidity of the portion of the spine that had been involved in the disease process. The motor and sensory power of both legs was normal and the patient was able to get about and work as before her illness.

Comment. It is a question whether this patient would not have recovered from the paraplegia without operation had the condition been treated by recumbency and hyperextension of the spine from the time it was diagnosed, which was shortly after the onset. The cause of the paralysis was compression by bone, a condition usually relieved by hyperextension. In view of the peculiar shape of the bone angulation, it is impossible to say that hyperextension would have helped this particular patient. However, it was not used and at the time I saw the patient, eight months after the onset of the disease, the paralysis was severe and rapidly becoming worse. Because of this we decided it was unwise to delay operation in order to attempt to secure recovery by conservative treatment. The result showed that this decision was correct. I wish to emphasize this, because it is a situation the surgeon will frequently have to meet. Since most of the paraplegias of spinal tuberculosis recover with conservative treatment, it is the usual practice to persist with these methods for many months after a time when the lack of improvement, and sometimes steadily increasing impairment, make it evident that some other method of treatment is necessary if we are to relieve the cord impairment. In no other spinal condition is it good treatment to allow a compression paraplegia to persist, unimproved or growing worse, over a period of many months without an attempt being made to relieve the condition by exploration. And the paraplegias of spinal tuberculosis should not be held to be exempt from this rule, after proper conservative treatment has failed in six to eight months.

CASE III. N. W., male, 12 years, was a normal healthy child until March, 1916, when he stumbled and fell while walking. He complained of pain in the left leg, but was able to walk after a few minutes. A week later he began to limp on the left leg and complained of pain in the thoracic region of the spine. Two weeks later a small protrusion was noticed on the back at the level of the eighth thoracic vertebra. There was gradual loss of power so that six weeks

after the onset the left leg was partially paralyzed and the right leg showed marked weakness. By July 1, 1916, three months after the onset, there was complete paralysis of both legs, but no impairment of the sphincters. Tuberculin and Wassermann reactions at this time were negative. Because of the negative tuberculin reaction and the indefinite roentgenograms his physician ruled out tuberculosis, and made a diagnosis of extradural cyst or sarcoma.

Status, September 8, 1916. A poorly nourished boy with complete paralysis of both lower extremities up to the groin, the legs spastic with exaggerated knee- and ankle-jerks. Ankle- and patellar clonus present on both sides and double Babinski sign present. Touch sense was impaired in large patches over both legs with slight hypesthesia over the abdomen below the umbilicus and marked diminution of pain and thermal sense over both lower extremities to the groin. He complained of shooting pains in both legs below the knee. The sphincters were unimpaired. Roentgenograms showed no evidence of disease in the vertebral bodies, but there was an indefinite blurring about the spinal arches of the sixth, seventh and eighth vertebrae. A tentative diagnosis of either extradural sarcoma or spinal tuberculosis was made, and because of the evidence of severe cord involvement, operation was advised.

Operation. Laminectomy, September 9, 1916, through a midline incision extending from the fourth to the eighth thoracic spines. Upon retracting the muscles on the left side a large cavity was disclosed, with marked bone destruction involving the left laminae, transverse processes and pedicles of the fifth, sixth and seventh vertebrae. The spinous processes and laminae of these vertebrae were removed with roenguers, disclosing a large mass of necrotic bone fragments and tissue overlying and compressing the dura. This was easily removed, the dura rounding out and pulsation returning to the compressed portion. The dura was smooth and glistening and showed no evidence of having been invaded by the disease process. The dura was not opened. The wound was closed in the usual manner, using two drains, and a dorsal plaster splint applied.

Post-operative. Convalescence was rather prolonged but there was some improvement in sensation eight days after operation, touch sense showing the greater gain. There was some return of motor power two weeks later and when the boy left the hospital one month after the operation, he could weakly move both legs. Improvement was rapid after this, so that three months after the operation, sensation was normal and there was almost normal power in the legs. He wore plaster jackets and a back brace for eighteen months. Roentgenograms taken some weeks after the operation showed disease foci in the bodies of the fifth, sixth and seventh vertebrae. These had been obscured in the earlier pictures by the larger blurring posteriorly. A letter from his physician five years after the operation says that the boy is well, with normal strength in back and legs.

Comment. There are several points of interest in this patient's condition. It is rather rare to find tuberculosis attacking the spinal arches. It is usually not difficult with the aid of the *x*-rays to make a diagnosis of spinal tuberculosis. We were misled in this instance because of the rarity of the disease in the spinal arches, and because the vertebral bodies were apparently not involved. As a matter of fact they were diseased but the small foci present were obscured by the large mass posteriorly. The large pieces of necrotic bone and the coarse nature of the detritus in the canal that formed the mass compressing the cord make it extremely probable that it would have required a long period of time for its removal by absorption. Without operation it is hardly likely that the cord could have endured the severe compression over a long period of time without permanent damage. It was fortunate for this particular patient, that the diagnosis of tuberculosis was not made prior to operation, for had it been, perhaps we would have advised conservative treatment at least for a few months and the result probably would not have been so successful.

The prompt healing of the wound and the rapid improvement in the patient's condition supports the statement made earlier, that laminectomy for an impaired cord in Pott's disease is not dangerous, provided one confines his attentions to the cord and the canal, and does not attempt to interfere with the disease process in the vertebral bodies.

SUMMARY.

Involvement of the cord occurs in 5 to 8% of patients having tuberculosis of the spine. Ninety per cent. of the cases of cord impairment with paralysis will be wholly or almost wholly cured, if treated by recumbency and hyperextension of the spine on a vex stretcher frame. This treatment must frequently be carried out over a period of many months to remove the paralysis, but some signs of improvement should be present at the fourth month, certainly at the sixth to tenth month, if the treament is to prove successful.

Abscess is by far the most frequent cause of cord compression, and by far yields the best results with hyperextension. Compression by bone is second in order of frequency, and usually yields good results with hyperextension. The ten per cent. of paralyses persisting with this treatment are due to continued bone compression, constrictions in the dura, pachymeningitis and obliterative arteritis.

If there is no improvement in the paralysis in from six to ten months laminectomy should be considered. Naturally, one's decision as to operation will be influenced by the patient's general condition and the extent and severity of the paralysis. Compression by bone, detritus in the canal, constrictions of the

dura, can be relieved by laminectomy. Lesions within the dura will not secure much if any relief from operation as the dura should not be opened if the disease process is still active. If the paralysis persists when the spinal caries is cured, the cord can be explored by a second operation. If then the condition is found to be adhesions within the dura or a collection of fluid, much good may result. Severe pachymeningitis or myelomalcia resulting from an obliterative arteritis will not be helped.

By no means should laminectomy be performed indiscriminately in the paraplegias of Pott's disease. As stated repeatedly before, ninety per cent. of the patients with cord involvement will be relieved by recumbency and proper hyperextension. Laminectomy should be employed only in those cases that fail to show any improvement in the paralysis after six to ten months of treatment by hyperextension. But when thus indicated laminectomy should be performed promptly, and the patient not be permitted to drift along for years with a persisting paraplegia, until the degenerative changes in the cord render intervention hopeless.

HOW SHALL WE TREAT GALL-BLADDER DISEASE?*

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Gall-bladder disease is second to appendicitis as a cause of intra-abdominal morbidity, as shown by operative and autopsy records. In the latter the percentage of gall stones ran from five to six[1]. Many are but beginning to appreciate the frequency of cholecystitis with or without stones. In patients operated upon for gynecological conditions at the Mayo Clinic, and by Peterson of Ann Arbor, the incidence of gall stones was 7.2% and 12.5%, respectively, in several thousand cases. Naunyn even goes so far as to say that ten per cent. of people have gall stone disease. Cholecystitis without stones is, undoubtedly, of a frequency equal to that of gall stones.[2] For too long a time gall-bladder disease has meant, to the profession, gall stones and a failure to palpate stones has erroneously given the gall bladder a clean bill of health. Until gall stones are considered only an incident in the pathological process, there is little hope for further improvement in the result of our surgical therapy. Graham, of Toronto, has well expressed this by saying—"To wait for jaundice and colic in order to make a diagnosis is like waiting for metastases before diagnosing a malignant growth".[3]

*Read before the Cincinnati Academy of Medicine, January 9, 1923.

There has been a steady change of opinion in gall-bladder surgery in recent years; today, it can be safely said the weight of opinion is in favor of cholecystectomy rather than cholecsytostomy. I know of no surgeon who, having accepted cholecystectomy, has gone back to cholecystostomy as the operation of preference. Early surgery of the gall-bladder was done largely to conserve life in advanced stages of disease. A few years ago, removal of a gall-bladder was the exception and performed only on unusual evidence, at operation, of very advanced disease. In this way, many cases of cholecystitis remained undiscovered and uncured and many stones were missed. The presence or absence of stones is frequently impossible of proof, unless the gall-bladder is opened, and if this precaution is not taken, inflammation may be and new growths are almost sure to be overlooked; exploratory cholecystostomy is not a wise procedure. As a general statement, it would seem that a gall-bladder needing surgical intervention had best be removed, unless there be some positive contraindication, as will later be mentioned.

As skill, knowledge, technic, and early diagnosis have developed, mere drainage of appendiceal abscesses has steadily become less frequent, until today this procedure is the exception. Today we rid the body of the diseased appendix as well as its products. It is fair to assume that we should do likewise in the case of the diseased gall-bladder, and not merely remove the stones and thickened bile. Gall stones are the ashes of the fire. Lagenbuck[4] said in 1882, "Gall-bladders should be removed not because they sometimes contain stones but because they make stones"; and another surgeon expresses it, "The operation is not done for the purpose of removing stones, for these may or may not be present, but for the purpose of removing tissue diseased as a result of progressive inflammation".

Drainage of the gall-bladder, either surgical or the recently devised method of so-called non-surgical drainage, is based upon the premise that such drainage will sterilize the gall-bladder. Unquestionably, there are examples where drainage is the method of choice for even the experienced surgeon, but even in these cases, would it not be wiser to advise a secondary operation for the removal of the gall-bladder, as is customary, in those now rare cases of simple peritoneal drainage of peritonitis, complicating appendicitis? From the beginning I have been opposed to the principle of medical, or non-surgical drainage, of

the gall-bladder as a means of treatment of all but a very small percentage of these cases; it is not a logical procedure because it does not and cannot properly drain these gall-bladders. The catarrhal jaundices fall in the group that may be drained non-surgically with real benefit. Who has ever seen a gall-bladder contract or nearly empty as a result of an automatic process? Every surgeon who has tried to express bile from a gall-bladder knows how much pressure is required to accomplish this, even when he has pronounced the gall-bladder and ducts normal. The few non-striated muscle fibers in the extremely thin wall of a gall-bladder cannot be expected to exert much force toward accomplishing the complete, or partial, evacuation of the bile contents; and one is led to believe the gall-bladder is a decidedly inactive organ, that takes little part in the storage, expulsion, or essential modification of bile.[5] In a thickened, inflamed gall-bladder the contraction of the tiny muscle fibers must be impossible.

The advocates of non-surgical drainage say the duodenal bulb should be left in place for the purpose of draining the biliary system, an hour or so at a time. How many patients will consent to daily treatments of this length? If these are permitted, then this method claims to facilitate drainage of the gall-bladder one twenty-fourth of the time the patient is under treatment. Surgical drainage, which is through and through drainage, and complete twenty-four twenty-fourths of the time during the drainage period of fourteen to twenty-eight consecutive days, is an acknowledged failure in a definite percentage of cases. Therefore, a method of drainage acting, at most, one twenty-fourth of the time must fail to sterilize, or permit auto-sterilization, of the mucous membrane lining containing thousands of crypts and mucous gland mouths.

In the above argument it has been presupposed the duodenal tube method actually drains the gall-bladder; it does not even do this for it has been shown the gall-bladder, cannot forcibly contract and completely empty itself.[6 7] An outflow from the gall-bladder must of necessity be intermittent, as there is but one passage for entrance and exit of the bile; the bile cannot flow both ways in a tube two to three millimeters in diameter. In a case of cholecystitis, some infected bile flows out, let us assume, under the stimulus of the ounce of magnesium sulphate deposited in the neighborhood of the papilla of Vater; some remains in the incompletely contracted gall-bladder; bile from the liver sooner or later flows in and mixes with the infected gall-bladder contents. This may be repeated indefinitely but will there ever come a time when the original infected bile can be said to have been drained out completely? And we have made no allowance for the tremendously rapid bacterial reproduction. The chief advocate of this duodenal tube method says it is not to be used in cases of gall stones. How is one to differentiate the cases with stones? Surely not by the colic symptoms, for many cases of gall stones are being treated daily by this method, and as an extreme instance of this fact, I operated upon a woman about a year ago who had had thirty-eight "trans-duodenal gall-bladder taps", during some three months of continuous jaundice due to a large common duct stone. Only recently I saw in consultation, a man who had been "jaundiced" for twenty-five years, i.e., since fourteen years of age, and upon whom two bile tract operations had been performed; his condition is that of "hemolytic jaundice". Needless to say, this man was not a case for bile tract surgery or drainage, but he had had one hundred duodenal "non-surgical taps". He needed a correct diagnosis. These instances are not cited in a spirit of criticism, but to point out the result of this illogical method of treatment, and the extremes to which a faulty premise make take one. The method has become a fad; and I prophesy that two more years will see the completion of its passing, now begun, as have passed other fads illogically founded, e.g., anoci association. The great good that has come out of the Lyon-Rehfuss tube method of treatment is a better appreciation of the widespread incidence of gall-bladder disease.

Reporting the meeting of the Southern Medical Association, November, 1922, the *Journal of the A. M. A.*, p. 2110, 1922, quotes Dr. Martin E. Rehfuss, as follows:

"One always ought to be on guard for possible associated diseases, of which the most common are chronic appendicitis, chronic colitis, and chronic lesions of the stomach and duodenum. It is my rule to urge operation when medical treatment fails to produce amelioration of symptoms, and fails to produce a cessation of the acute attack; when there are gross deformities in the upper right quadrant, usually demonstrated by the roentgen ray; in the presence of acute inflammatory diseases; in the presence of common duct block, or in the presence of chronic gall-bladder disease during the malignancy stage." Very few follow the instructions thus laid down.

Examples of conditions where surgical drainage may be preferable to excision are: deep jaundice with associated stone in the common duct; cases of acute, or chronic, cholecystitis with or without stones

and surrounding adhesions, so acutely ill that the time element is of greatest importance; those rare cases in which one has strong reason to suspect an infection of the hepatic ducts; and a few cases where the gall-bladder is needed for cholecystenterostomy. Some go so far as to say, even these gall-bladders should be removed and the drainage obtained through the common duct or cystic duct. Personally, I have also felt my limitation in one other group, where the physical construction of the individual, is such as to make the exposure of the ducts very difficult and unsatisfactory. Some of the short-waisted, fat, deep-chested people fall into this class, particularly if the under surface of the liver slopes at an acute angle up behind the costal margin, and the several aids to exposure, described in the literature, fail to accomplish the desired result. In the above types, the higher percentage of cures following cholecystectomy is not great enough to warrant large additional risk. The fact that cholecystectomy is a better operation is no serious reflection upon the value of cholecystostomy, for even here the percentage of good results is high, considering the widespread ramifications and seriousness of the disease in the late cases. Drainage, rather prolonged, should be the operation of choice for the occasional operator. Experience and inexperience are relative terms, for no surgeon is so good that he cannot be better, to the benefit of his patients.

Drainage for several weeks may fail of sterilization of an infected gall-bladder because:

(1) A gall-bladder is so constructed that it has never been known to completely empty itself,[6 7] and, therefore, stasis, residual bile and continued infection frequently result. When one speaks of an infected gall-bladder, the walls of the gall-bladder are referred to and not merely the contents; the infection is interstitial.

(2) The many thousands of secreting glands and crypts in the lining of the gall-bladder are not approachable for attack in an effort to disinfect them, and they become retention pockets of sediment, pus, and bacteria. It is extremely difficult to sterilize a urethra which is flushed with several quarts of fluid, at frequent intervals, while a gall-bladder is merely filled by a back-flow and is never flushed through.

(3) Long continued drainage, that is, fourteen to twenty-eight days of twenty-four hours each, necessitates the continual presence of a drainage tube in the gall-bladder where it may act as a foreign body producing irritation with more or less of reactive changes, even to perforation.

Those still in favor of cholecystostomy base their argument on three postulates: first, that the gall-bladder has a function necessary to the smooth operation of the human economy; second, removal is a more serious operation than drainage; third, should trouble develop in the common duct later, the presence of a gall-bladder is desirable as a means of anastomosis to the duodenum or as a landmark to locate the ducts.

Some of the functions ascribed to the gall-bladder in recent literature are that it:

(a) acts as a tension bulb, or similarly to a steam regulator on a steam engine, and so equalizes the intrabiliary system pressure.

(b) stabilizes the flow of bile through the common duct.

(c) is for the purpose of bile storage.

(d) is for the purpose of condensing the bile, thus increasing the storage capacity of the organ.[8]

(e) dilutes the bile with mucin, thus making the bile less irritating.

These claims for a function of importance can be easily answered:

(1) The thousands of instances in which the gall-bladder has been removed, without any known unpleasant sequelae, is positive evidence as to the small importance of any function, if there is a function.

(2) To what extent bile is an excretion or a secretion, or both, remains to be ascertained, but the excretory function of the liver is, to say the least, important, and it is illogical to assume that the body economy would permit of storage and concentration of a poisonous excretion. The concentrating action of the gall-bladder, as demonstrated by Rous and McMastian, can well be questioned in most cases of cholecystitis as the mucous membrane of the gall-bladder is, to a more or less great extent, destroyed in this condition.

(3) Comparative anatomy indicates that the gall-bladder has no function, for of animals with similar habits, some have and some have not gall-bladders; for example, it is absent in the deer, horse, rhinoceros, ass and pigeon, while present in the cow, sheep, hog, hen and turkey.

(4) The gall-bladder storage capacity is equal to the amount secreted in about one-half hour by an actively functioning liver. The remaining portion of the bile secreted must go direct from liver to intestine, when needed, in answer to the hormonic stimulus. Thus a large part of the bile is normally not subjected to the beneficial influences of the gall-bladder habitation and modification that is made a point of by some, in an attempt to substantiate a theory of gall-bladder function, namely, that the nucleo-protein mucin of the gall-bladder lessens the toxicity of the bile.

The second objection to cholecystectomy is sound;

it may be more serious, as greater harm can be done, accidentally. Cholecystostomy is undoubtedly more nearly "fool-proof".

Answering the third objection; it is believed, and by many thought to be, an established fact, that less trouble follows excision than drainage of the gall-bladder. There is no doubt, however, that the presence of a gall-bladder is an aid, should a second operation be necessary, but *fewer second operations will be necessary*. Deaver [9] says that 65% of his recurrences could have been obviated by removal of the gall-bladder, and Brewer[10] says that cases of stones recurring in the common duct are strikingly fewer, after cholecystectomy, than those after cholecystostomy. If the following reasons for cholecystectomy are true, then less morbidity will follow it:

(1) The removal of diseased tissue is, today, the object of the surgical procedure, provided, that, proven harm is not done thereby.

(2) Cholecystectomy, by the very nature of the operation, more fully insures the removal of the stones, and at the same time makes possible a more thorough examination of the ducts.

(3) A gall-bladder once infected is usually permanently infected and damaged, and a recurrence of symptoms is apt to follow. Here is the heart of the cholecystectomy argument. We see many times, clinically, and the laboratory confirms it, that a gall-bladder wall once infected retains its infection indefinitely, so that inflammatory processes are frequently recurrent. Through the lymphatics and by continuity of tissue these infections may be spread to the liver, pancreas, and other organs. The gall-bladder becomes the focus of infection for the entire digestive tract and body, just as easily as a tonsil becomes the source of infection for a distant arthritis or a neighboring peritonsillar abscess. So long as the gall-bladder continues to be a depot of infection, the liver, pancreas and allied organs cannot functionate normally, both because of disturbed reflexes through the sympathetic system, and because of fibrotic changes that may be instituted in these organs. Remove the gall-bladder and these pathologic processes are interrupted and the parts may return to near normal, just as one sees the subsidence of an arthritis after tonsillectomy.

(4) During a drainage operation, soiling of the peritoneum is more apt to occur with local peritoneal adhesions as a sequela.

(5) After operation, leakage around the tube used in cholecystostomy will occur more frequently than after cholecystectomy.

(6) Pre-operative adhesions to the diseased gall-bladder are sure to reform after operation.

(7) Peritoneal adhesions are almost constantly present after gall-bladder drainage and their extent and severity are often out of all proportion to the primary affection and the nature of the operation. Bile has been shown to be very irritating to the peritoneum.[11] Adhesions to a drained gall-bladder may so anchor it that biliary stasis will commonly follow, with its train of events of bile-salt and cholesterin precipitation, and secondary stone formation. The evidence of reformation of stones is complete. Nine and five-tenths per cent. of drainage cases, in the hands of forty-five of America's most experienced surgeons, had a recurrence of symptoms.[12]

(8) Cholecystostomy leaves the fundus of the gall-bladder attached to the abdominal wall, either intentionally by suture of the surgeon, or accidentally, by scar tissue along the drainage sinus. This cannot help but distort and pull upon the gall-bladder, in many cases producing, at least, a dragging soreness in the wound.

(9) Adhesions to a drained gall-bladder or its duct may distort these so that the difficulty of a free to and fro flow of bile is increased.

(10) A thickened gall-bladder wall means a narrowed cystic duct, normally only two or three millimeters in diameter, and long drainage may contribute to its further narrowing rather than dilatation.

(11) The preponderance of ulcers within the few inches of the duodenum as compared with the more than twenty feet of gastro-intestinal tract, may be, in part, accounted for by the close proximity of the point of outlet of infected gall-bladders, typhoid sequelae, pneumonia, acute and focal infections, etc.

(12) Convalescence is not only more smooth following cholecystectomy, but also less disagreeable because of the abscence of drainage bottles, frequent dressings, etc.

(13) Excision of the gall-bladder has been proven, experimentally, to lessen the resistance of the sphincter of Oddi to bile flow, and lower the intra-ductal pressure from 100 to 150 millimeters of water down to about 30 millimeters of water. Clinically, this may well be true as it explains the improvement after operation of the cases of pancreatitis complicating gall-bladder disease.

(14) Failure to remove a gall-bladder at the primary operation is a cause of 90% of biliary fistulas.[13]

(15) Excision removes a not uncommon site of cancer, which has been found in as high as 4.5% of a large series of gall-bladder operations.[12]

(16) Last, and of lesser importance, but still valuable, is the fact that patients are in a better frame of mind. The medical profession has so stressed the

idea of "removal of diseased tissue" that the layman feels better satisfied when the diseased gall-bladder is removed. Not a few have expressed themselves to me, "I'm glad you took it out", or I'm sorry you did not take it out; what will prevent stones forming again?"

The immediate pathologic indications for removal of the gall-bladder may be divided into two groups:

First, the group that all surgeons are pretty well agreed upon as being good reasons for cholecystectomy:

(1) Early suspected primary cancer of the gall-bladder.

(2) Gangrene of the gall-bladder.

(3) The atrophic shriveled-up gall-bladder.

(4) The functionless gall-bladder that has thick inflamed walls.

(5) Empyema of the gall-bladder due to stricture or stone impacted in the cystic duct.

Second, a group over which there is a difference of opinion:

(1) Gall-bladders adherent to neighboring more important organs as the stomach, duodenum, colon, etc., when there is no demonstrable lesion in these latter organs to account for the adhesions.

(2) Gall-bladders of normal external appearance containing one or more stones.

(3) Chronically inflamed or infected gall-bladders with or without stones. The strawberry gall-bladder, so-called, falls in this group.

(4) Acutely inflamed gall-bladders, primarily or secondarily to chronic inflammation.

Bile tract surgery carries a mortality directly in proportion to the severity of the complications, and where the disease is mostly confined to the gall-bladder, the mortality is about one to one and a half per cent. The lesson to be drawn, is that a gall-bladder should be surgically dealt with at a time when the mortality is low, the disease largely confined to the gall-bladder, and a secondary complication of duct obstruction, hepatitis, pancreatitis, adhesions, perforation, liver abscess, nephritis, myocardial changes, etc., as yet undeveloped. Waiting for the classical, text-book, localizing signs and the above-mentioned, far-reaching, secondary lesions, gives an immediate surgical mortality of from 20% to 30% and a small percentage of cures in the survivors. The deaths most often occur from cessation of liver function, the result of infection and damming of bile, or exhaustion from blood changes due to chronic cholemia.

It was the alarming mortality resulting from the complications of prolonged medical treatment and delayed surgery, that placed the early prompt operation for appendicitis on its present sound surgical footing. Late operations mean high mortality. Do not permit high mortality to mean late operation, provided delay is the cause of the high mortality.

Gall-bladder disease, a result of bacterial invasion in childhood or early adult life, has been, in the past, progressive for years, until unbearable pain, the yellow flag of jaundice, or the cachexia of malignancy drives the patient to the surgeon. The surgery of the gall-bladder has been twenty years behind the disease. The future of gall-bladder surgery lies in an early attack upon and eradication of the gall-bladder as the focus of widespread infection.

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THE SURGERY OF SPLENIC ANEMIA.

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I. HISTORY AND NOMENCLATURE.

Speculation concerning the function of the spleen has probably exceeded that of any other organ. It has been called the organ of mystery, the organ of romance! Because of our lack of knowledge the lure remains in spite of refuted evidence, disappointment in the experimental aspects and discouragement in the face of our inability to recognize more than a few facts concerning the physiology of the organ. In health and in disease its most important relationships are to the blood.

The spleen is one of the main filtration plants of the body. Worn out blood cells, infectious and toxic agents are separated in this organ from the blood stream, and passed on to its neighboring collaborator the liver, for detoxication. The spleen may be overpowered by the accumulation of the residue of bacteria, or toxic substances which are unfiltered or, in the presence of an otherwise incompetent spleen, isolation of these products may occur within the organ as the result of failure to pass them on to the liver. The spleen enlarges and represents a defensive, or-

ganic reaction to these noxious agents. It is no longer capable of ridding itself of its unwelcome guests. When the spleen loses this function and allows the infected and toxic blood to escape the filter of its sieve-like action, the liver which has already been receiving bacteria-laden and toxin-laden blood receives the full brunt of the poisoned portal circulation and finally itself succumbs to cirrhotic changes.

Many of the older myths have been dispelled. The most important fact of value in treatment is that in some way compensation is afforded for the loss of the spleen after its successful removal and provision is made for the continuation of splenic functions through vicarious organs or other members of the blood-forming system.

Virchow first associated abnormal blood states with enlargement of the spleen and his discovery of leukemia in 1845 (claimed by Moynihan for Hughes Bennett) paved the way for further knowledge of its diseases. Later Cohnheim separated another group of the splenomegalias into the pseudoleukemias.

The term splenic anemia was first used by Gretzel to designate a case occurring in a child of what we now know as *von Jaksch's anemia* (*infantile pseudoleukemia*). Other terms applied have been *splenic pseudoleukemia* (Eichorst) and *primary splenomegaly*. (Debove and Bruhl). In 1883, Guido Banti, of Florence, added confusion to existing chaos when he described that form of primary splenic enlargement bearing his name.

A few early writings by Woods and others threw light upon the subject, but Banti deserves the most credit for bringing the disease before the profession. The late Sir Wm. Osler, who contributed much to the subject, considered primary splenomegaly and so-called *Banti's disease* initial and late stages, respectively, of *splenic anemia*.

Banti contributed his three important papers, in 1883, 1894, and 1910. In 1894, Banti described three ill-defined clinical stages which merged into each other. The first stage, according to him, was characterized by splenic enlargement with anemia, which lasted three-twelve years. In the second stage, of brief duration, the liver became enlarged and the quantity of urine diminished. In the third stage, the liver shrank gradually in size, ascites appeared with the symptoms of atrophic cirrhosis and the patient invariably died in one or two years from hemorrhage or auto-intoxication. In 1910, Banti attempted to ascribe a definite pathological picture to the disease as follows: 1. Fibrosis of the Malpighian follicles and reticulem of the spleen. 2. Narrowing of the splenic veins, thickening of the splenic capsule and trabeculae. 3. Endophlebitis with calcification of the splenic veins extending up to the portal vein. 4. Cirrhosis of the liver of the interlobular type of Laennec.

Types of anemia in infancy and childhood associated with splenomegalia have been described. These have given rise to added confusion. Gretzel, first described in 1866 a group occurring in children and suggested the name "splenic anemia of infancy." Attention was re-called to this by Somma in 1884. A few years later von Jaksch directed further attention to an anemia with leucocytosis and enlarged spleen but adopted an unfortunate name, *anemia pseudo-leukemica infantum*.

The disease was analysed further by Hunter in 1909 who described 10 cases of *infantile splenic anemia*. According to this author the two diagnostic criteria are anemia and splenic enlargement. He divides the disease into three groups, characterized as follows: 1. There is a decrease in the number of leucocytes in which lymphocytes preponderate. 2. A moderate leucocytosis up to 20,000 and a few myelocytes. 3. A greater increase in the number of leucocytes, this type conforming to von Jaksch's anemia.

There are two chief forms of splenic anemia which are found in infancy and childhood to which Giffin draws attention: type I, which conforms to that which occurs in the adult; and type II, now generally known as von Jaksch's anemia. For the present, until our knowledge is more complete, Giffin believes it is best to draw a distinction between the two conditions. There has been a decided lack of standard classification in these forms. The chief difference between the adult form of splenic anemia and the splenic anemia of infancy is in the blood picture and more particularly in the leukocyte counts. In the splenic anemia of infancy there is also more evidence of blood destruction than in the splenic anemia of adults. The red-cell count is likely to be lower and the color index consequently higher, normoblasts and megaloblasts are present in the blood smears and there is a notable leucocytosis (lymphocytosis). In splenic anemia of the adult type occurring in children there is an absence of leucocytosis. Just what determines a leucopenia in the one and a leucocytosis in the other it is difficult to state, but our knowledge concerning the normal blood of infants and its behavior to toxic agents would lead us to regard the form attended with leucocytosis as a less surprising reaction. Childhood is a transition period, in which there may be a reversion to the fetal type of blood formation and this fact may have an important bearing upon the variations in childhood.

Tropical splenomegalia, or *kala-azar*, recognized since about 1869, is marked by progressive anemia and emaciation, enlargement of the spleen and liver, dropsy and intestinal ulceration. The disease resembles splenic anemia closely in many respects and occurs in both adults and children. A parasite has been isolated, the Leishman-Donovan body, which is now considered the specific cause of the disease. It infests the endothelial cells of the spleen, the liver, the bone-marrow and less often the other tissues and organs.

Ponos has been considered an infantile form of splenic anemia with fever, endemic in the Greek islands of Spezzia and Hydra. It is now considered clinically indistinguishable from a form of infantile splenic anemia due to Leishman-Donovan-like bodies later described by Pianse in 1905. However, cases so far described of the Pianse infection have been limited geographically to Sicily, the Liparic Islands, Calabria, Tunis and Malta. The morphology of the

Leishman-Donovan parasite of kala-azar is identical with the Pianse organism found in certain infantile types of splenic anemia, but it presents certain other marked differences. Further investigation of the cause of ponos may show that it is an infection identical with the Pianse organism. It is plain that these infantile anemias with fever and splenomegalia, i.e., kala-azar, the type due to the Pianse organism, and ponos are intimately related clinically and pathologically and probably etiologically.

Sporadic cases of anemia with enlargement of the spleen, known as *splenostomachon,* are met in several parts of the mainland in Greece, and like the splenic anemias of infancy elsewhere, are generally associated with rickets, congenital syphilis or malaria. The most striking peculiarities of that form of splenic anemia distinguished as ponos are its restrictions to and endemic prevalence in the two small islands of Spezzia and Hydra, its sudden and comparatively recent appearance, and its great fatality.

II. Etiology and Pathogenesis.

Banti deserves credit for his attempt to elucidate the mode of origin of the disease which bears his name and for awakening interest in these problems. He at one time assumed that an unknown toxin, developed in the fibrotic spleen, which is itself caused by an unknown toxin, in turn later causes secondary cirrhosis of the liver. Mallory considered that he had demonstrated that a spleno-toxin may cause certain changes in the liver. After extensive trauma of the spleen he found focal necrosis in the liver. Opposed to this view are the facts that dead tissue is not a toxin, that focal necrosis in the liver is not nor can it produce cirrhosis, that the production of a toxin in one organ which in turn produces secondary changes in a distant organ has no analogue in pathology. The first sclerosing agent usually produces lesions simultaneously.

The discussion as to whether splenic fibrosis is primary, as maintained by Banti, or of inflammatory origin, is not of great importance. Banti held that because he did not observe fibroblasts in the spleens of the disease bearing his name that the process is primary. As Eli Moschcowitz points out, however, cells resembling fibroblasts are seen in these spleens. Chiari, who in part share Banti's views, is one of many, however, who holds that fibrosis of Banti's disease is identical with splenic fibrosis of other origin.

Moschcowitz believes that a fibrogenetic toxin, probably of intestinal origin, attacks the organs draining the portal area and causes primarily a fibrosis of the spleen. If the toxin is sufficiently intense or the patient lives a sufficiently long time, it also causes cirrhosis of the liver as well. He believes that sclerotic vascular changes in the mesenteric vessels of so-called Banti's disease are to be explained on the same grounds.

Banti's latest written opinions favor some infective agent and the belief that the spleen produces another toxin which acts on the liver and splenic veins. It is pointed out by Moynihan that this is not only unnecessarily complex but has no parallel in the production of disease for there is no reason why a single agent could not produce both splenic and hepatic lesions. The significant fact that splenectomy leads to a cure or arrest of the disease certainly forces the view that the spleen plays an important rôle in the disease. From this fact we must assume one of two alternatives, that the disease is either primarily located in the spleen or that the spleen modifies the action of some agent located elsewhere. If we assume the second hypothesis—and such a view is held by those who believe that splenomegalia is to be attributed to excessive hemolytic activity of the spleen—the explanation of the outstanding microscopic feature, fibrosis, is not apparent. If, on the other hand, it is held that the seat of the disease is essentially primary in the spleen, search for the microbe or toxin in the spleen should ultimately prove successful. In this connection it is urged that immediate bacteriologic examination of excised spleens be made to provide material for study on this point. So far these immediate-postoperative investigations have brought no definite findings to light. This failure has been explained by the possible disappearance of the infective organisms from the scene of their activity or by our inability with present methods to detect them or the toxins.

Hollins holds that the colon bacillus accounts for the fibrotic changes in the spleen and liver. Moynihan suggests that the spleen harbors various organisms. Some excite local fibrosis in that organ, others in the liver.

Is anemia an essential part of the disease or is it incidental? It is plain that the resulting anemia may be due to toxemia or to hemorrhage. Probably in the beginning it is due to toxemia in view of the usual leucopenia attending the disease. Hemorrhage no doubt modifies the blood picture of the leucocytes, substituting a leucocytosis or at least some modification in the leucocytes from the usual leucopenia, and is to be held to account for the varying conditions occasionally seen in splenic anemia.

Rolleston's view that anemia is due to hemorrhages does not necessarily follow simply because some degrees of regeneration occurs after these attacks of bleeding. The mechanism of blood regeneration is not completely destroyed in splenic anemia and there is no reason to believe that regeneration should not take place. In addition to this, anemia may be present independently of hemorrhages. Hollins believes that colilysin, a toxin of the colon bacillus, produces anemia.

The cause of hemorrhage may be mechanical or toxic. Rolleston's reasoning is rational in ascribing hemorrhages into the stomach to mechanics—from the vasa brevia distended as the result of torsion of the splenic vein caused by the bulk of the spleen.

Hemorrhages may occur so massive that it is certain large vessels are involved. The relation of gastric hemorrhage is not entirely clear, but it is a significant fact that splenectomy has cured hematemesis associated with splenic enlargements of idiopathic origin, as shown by Balfour. It is possible that the less extensive hemorrhages are due to toxic ulcers. The only explanation may be that by splenectomy the portal circulation is reduced and back pressure lessened, but it seems more likely that a

combination of factors enter into the relief obtained.

As regards the infantile form of splenic anemia, Hunter believes all forms, including von Jaksch's anemia, are secondary to poor hygienic surroundings, poor food, syphilis, rickets and infectious diseases, especially measles. Naegli and Grawitz also believe this is a secondary anemia with certain usual features explained by the as yet unstable mechanism of blood formation in young children with reversion to fetal type. Many of these types in childhood are no doubt of known origin and are due to rickets, syphilis, and other causes. There still remains, however, as in the splenic anemia of adults, a group in which etiology is as yet obscure. Carpenter, in a review of 348 patients with splenomegalia under 12 years of age, places rickets first and syphilis second in the etiologic rôle. Ashby concludes that the toxin causing rickets may also cause the splenic anemia of infancy, and that the reported cases vary from those with marked bone changes and small spleen to those with slight bone changes and very large spleen. Carr, however, makes the following points: that in the majority of rickety children there is no splenic enlargement, that there is no connection between the severity of the rickets and the size of the spleen, or the degree of anemia, that in certain cases of splenic anemia of infancy there is no evidence whatever of rickets.

Referring now to the tropical form of splenomegalia, kala-azar, the transmission of the disease to man occurs by means of the bite of the bed-bug *cimex rotundatus*. To W. S. Patton is due the credit for tracing the extra-corporeal development of the Leishman-Donovan bodies, but to Rogers belongs the credit for first suggesting this mode of transmission. Rogers successfully cultivated the parasites in acidified, sterile blood withdrawn from the stomach of bugs that had been allowed to bite infected individuals. In a personal communication to Leishman, Patton stated that he had successfully infected bugs at Madras by allowing them to feed on kala-azar patients in whose peripheral blood Leishman bodies had been found. In the gut of such bugs he subsequently found flagellated bodies resembling those which develop in artificial cultures of the parasites. A detailed account of Patton's work has since been published.

The mode of origin of this disease seems to be as follows: A parasite, when introduced into the body, gains entrance into, or is taken up by the phagocytic action of an endothelial cell lining a small capillary or lymphatic, where the slowing of the current and the narrow caliber of the vessel favors such action. The parasite, however, is not destroyed by intracellular digestion within the endothelial cell but is able to live and also to multiply. This multiplication occurs by direct fission, and forms showing all stages of division can be seen in such cells. As multiplication goes on the cells increase in size, and the nucleus is often displaced to one side. In smear preparations from an infected tissue, in which the individual cell can be readily studied, as many as 200 parasites may be counted in a single cell. This process of cell infection can be best seen in sections of an infected liver in which the endothelial cells lining the portal capillaries are seen in all stages of infection and the gradual detachment of the infected cell from the capillary wall is well brought out. Subsequently, the infected cells may rupture, either through the tension caused by their burden of parasites or by reason of a degeneration of the protoplasm, and the parasites thus set free are at once taken up by fresh cells and the process of intracellular multiplication is repeated. The so-called matrix, in which small groups of parasites are frequently found imbedded, is no doubt made up of portions of the protoplasm of the cell that originally harbored them. The detached and infected endothelial cells are probably unable to move far by reason of their large size and possible loss of elasticity and power of accommodation to the narrow lumen of the finest capillaries, but the smaller cells or infected leucocytes are doubtless carried away in the blood or lymph currents. It is probably in this manner that infection is spread throughout the body. The occasional occurrence of the parasites in the leucocytes of the peripheral blood has been noted, but the rarity of their detection in this situation is remarkable in view of the intense infection usually found in the spleen, liver and bone-marrow.

As regards ponos, the tropical, infantile form of splenic anemia and another type of the disease described by Pianse, the identity of these conditions has not yet been accurately determined; but that they are intimately related clinically and pathologically to the adult type of infantile splenic anemia with leucopenia, and to kala-azar, is very obvious.

In 1905, Pianse first announced the finding in various organs of patients suffering with infantile splenic anemia, of organisms morphologically indistinguishable from the Leishman-Donovan bodies of kala-azar. This has since been confirmed and the accumulated data in an article by Jemma and Di Christina leave little doubt as to the accuracy of the observation. Cases in which these Leishman-Donovan-like bodies of Pianse are found are said to be limited geographically to Sicily, the Liparic Islands, Calabria, Tunis and Malta.

Ponos, endemic in the Greek Islands of Spezzia and Hydra, near the Peloponnesus, is clinically indistinguishable from a type described by Pianse and investigation may prove them identical. Parasites have been rarely found in the blood by Pianse and later workers and their existence here has been disputed. The organism inhibits the spleen and may be obtained by splenic puncture. Identity of this type with kala-azar is disputed. The following points, however, favor a common organism: 1. Identity in morphology and distribution of parasites. 2. Similar pathologic findings. 3. Splenic and hepatic enlargement. 4. Alterations in the bone-marrow and intestinal ulceration. 5. Chronic course. Opposed to identity are the following: 1. The organism of Pianse has been cultivated to indefinite generations on Novy-McNeal medium, while that of kala-azar, has been grown only on citrated blood. 2. The Pianse organism has been inoculated into dogs, with some difficulty, but the inoculation of the organism of kala-azar has not been successful. 3. The almost complete limitation of the disease in the Mediter-

ranean to children and the occurrence of kala-azar at all ages argues against their identity.

The cause and mode of origin of other protozoan types of splenomegalia, syphilitic and malarial, are too well known to discuss at this time.

Blanket Hypotheses. The hypothesis of Klemperer and Hirschfield that the spleen possesses an internal secretion which stimulates red blood cell activity in the bone-marrow, seems to have something to recommend it in the mode of origin of certain splenic lesions, by reason of its adaptability—*if* an internal secretion can be demonstrated. Thus in splenic anemia there would be a disorder of the internal secretion giving rise to a diminution in the erythroblastic activity.

In 1916 Wm. J. Mayo argued that an internal secretion in the spleen did not exist because splenectomy did not deprive the body of an important constituent and because it is not under complete control of the nervous system as evidenced by its scanty supply from Auerbach's plexus. This view was iterated in 1921.

It may be admitted, however, that the conception of pathogenesis based on an internal secretion has the merit of supplying a possible explanation of the advantages conferred by splenectomy in so many varying conditions. We must suppose that, as in the case of other internal secretions there may be disorder arising from deficiency or from excess of secretion (hyposplenism, hypersplenism) or from infective processes, or again from metabolic disturbances, the chief difference being that in the case of deficiency of the splenic secretion compensation is more readily established, possibly by reason of the existence of normal splenic tissue outside the limits of the main organ, or through the action of the bone-marrow or other members of the blood-forming system.

Lesions may also be explained in part by some agent that upsets the normal balance of a two-fold action of the spleen in regulating red cell production. Stradomsky has promulgated this regulator action of the spleen on the bone-marrow which he believes inhibits over-production of red cells on the one hand and stimulates under-production on the other. Should these influences fail to balance each other a riot could be conceived to follow which would promptly affect the blood and explain its varied pictures in splenic disease.

Relation of Diseases of the Spleen to the Intestine. Attempts have been made to incriminate the colon in diseases of the spleen, especially pernicious anemia and splenic anemia, but there is little evidence to show that causative agents of these diseases arise in the large intestine. Excised colons have shown no evidence of disease except conditions of atony, adhesions and dilatation.

Pallin assumes that so-called Banti's disease with ascites may take its origin in the intestine. He found moderate jaundice, a much enlarged spleen, moderate anemia of the secondary type and pronounced leucopenia in an unmarried woman of 28 who for two years had had digestive disturbances, with constant jaundice except for two brief intermissions. Abdominal pain occurred at times, occasionally very severe. There was no indication of hemolytic jaundice, but examination of the spleen on removal showed the findings typical of the second stage of Banti's disease. Eppinger has also described a form of pseudo-Banti's disease with both digestive disturbances and jaundice. The symptoms a year after the splenectomy confirm the assumption that chronic enteritis was responsible for the digestive disturbance and also for the changes in the spleen. After splenectomy, the jaundice, the anemia and the leucopenia disappeared, but the abdominal symptoms persisted. *(To be continued)*

THE TREATMENT OF CONGENITAL ATRESIA OF THE ESOPHAGUS.

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Congenital atresia of the esophagus doubtless occurs with much greater frequency than the 153 cases reported to the early part of 1923 indicates. Accounts of two, three, or four cases by single observers lead to the conclusion that thousands of similar malformations have either not been recognized or remain unreported.

This condition does not appear to have been successfully treated in any recorded instance. Weiss[1], in reporting three esophageal atresias with associated esophago-tracheal fistulae, frankly commented, "From a practical standpoint, the most important consideration in regard to this anomaly is the hopelessness of our present day method of treatment."

Since it is obvious that, unoperated, all of these infants must inevitably die, any conceivable benefit to be derived from surgical treatment fully justifies the attempt. It is also apparent that, to hope for success, some more satisfactory method of treatment, based on the structural nature and disordered function of the malformation, must be developed.

For the consideration of treatment, all cases may be classified into two general groups: first, simple atresias of the esophagus; second, atresias of the esophagus with an associated fistula between the lower segment and some portion of the respiratory tract, usually the trachea just above its bifurcation (figure 1). In the latter group the esophageal orifice has a patency which permits inspiration of air into the stomach, and regurgitation of irritating gastric secretions into the respiratory tract. It is this patency that affords the diagnostic symptom of continuous frothy accumulation in the mouth and upper air passages, and likewise after gastrostomy permits milk to gush through the lower esophageal segment into the trachea.

It is evident that, in the treatment of esophageal atresias, a distinction must be drawn between the minority of cases in which there is an uncomplicated atresia, and those with an associated esophago-respiratory fistula.

The logical procedure in cases of the first type is gastrostomy. Unfortunately, this relatively favorable group constitutes only about one-fifth of the cases encountered.

A variety of methods has been used, in an effort to relieve cases of the second type, with uniform lack of success. Simple gastrostomy has been tried repeatedly, but all the infants died from operative shock or were drowned in the milk which passed through the fistula. Jejunostomy, suggested by Demoulin in 1904, has proven equally futile. Following jujunostomy, while the milk is introduced below the stomach, gastric secretions stimulated by the process of digestion may still enter the respiratory tract.

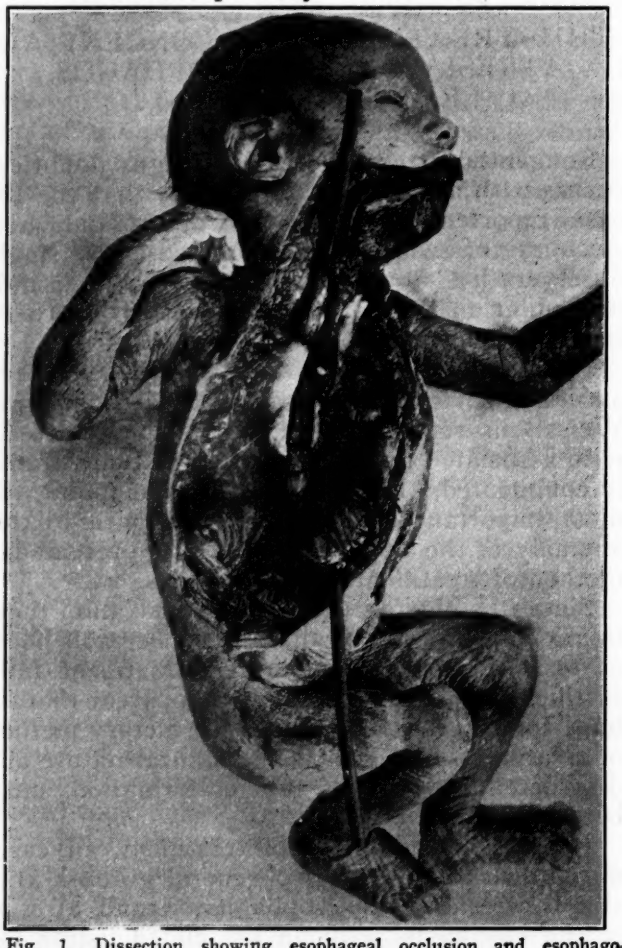

Fig. 1. Dissection showing esophageal occlusion and esophagotracheal fistula.

Richter[2] has used what appears to be the most reasonable procedure attempted to correct the conditions. In addition to gastrostomy, he made a posterior thoracotomy incision and, in two cases, closed the fistulous opening in the trachea. This daring maneuver, while ideal, has a rather limited application. It is doubtful if these feeble patients have sufficient strength to survive such a formidable procedure.

The closure of the fistula, even at a lower level than its opening into the trachea, seems essential before gastrostomy feeding can be attempted. While the following effort did not result successfully, we feel that the procedure is a logical one and deserves further trial and development:

R. R. B. Operation at Louisville Children's Free Hospital, April 18, 1921.

A short left para-median incision was made. At the suggestion of Dr. Karl David Winter, who assisted with the operation, a small blunt ligature carrier, loaded with coarse silk, was passed about the esophagus close to the stomach. The ligature was tied snugly, and the stomach replaced. Gastrostomy, using the Frank method to conserve time, completed the procedure (figure 2). Rectal injections of sodium bicarbonate, and intraperitoneal injections of physiological saline solution, were given to increase the alkaline reserve and to maintain body fluids. Breast milk was fed through the gastrostomy opening. Death followed in twenty hours.

As the abdominal esophagus is not entirely surrounded by peritoneum, effort should be made to pick up the lesser peritoneal sac in passing the ligature, which should be tied with only sufficient snugness to occlude the lumen. Spontaneous reestablishment of the orifice, and leakage, could be guarded against with greater certainty by crushing and invaginating the cardia where it has a posterior peritoneal covering. While this would also be an advantage, in case a subsequent effort should be made to establish the continuity

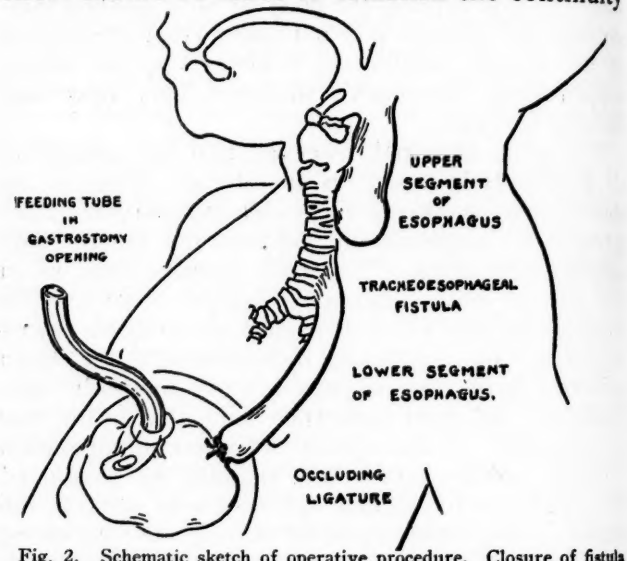

Fig. 2. Schematic sketch of operative procedure. Closure of fistula and gastrostomy.

of the esophagus, by leaving a pouch to which the stomach could be united, it would add materially to the severity of the operation and leave a small area of gastric mucosa above the occlusion to periodically expel its secretion into the respiratory passages.

Should one of these little patients live, then, several years later, through a left posterior thoractomy incision, proceeding with essentially the same technic employed in the two-stage operation for esophageal diverticula, the fistula could be closed and the two portions of the esophagus united. Subsequently the esophageal opening into the stomach could be reestablished. Closure of the gastrostomy opening would be a relatively simple matter. The carrying through of one of these cases to a successful conclusion would require the solution of a series of problems in technic, and the exercise of fine judgment, but the satisfaction of success would be immeasurable. Should the condition of the patient, or the judgment of the surgeon, decide against this rather formidable series of operations a continued gastrostomy life is conceivable.

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WALTER M. BRICKNER, B.S., M.D., F.A.C.S., *Editor*
New York City

ELMIRA, NEW YORK, JUNE, 1923.

SURGERY IN TOXIC GOITER.

In the department of "Progress in Surgery" in this issue of the JOURNAL are abstracts of articles upon thyroid surgery that have appeared within the past few weeks from the pens of men of large experience in this field. Taken collectively, they strike a new note or, rather, they put forward with the fresh assurance of competent observation, large material and careful critique, the value of surgery in a large group of cases of "toxic goiter", including both those of the exophthalmic type and those that have come to be classified as "toxic adenomata."

It must be said that there has been, and with much reason, considerable skepticism, not only among internists, but also among surgeons, concerning the classification of toxic goiter as a surgical disease; and, admittedly, surgery has had to bear the blame for many unsatisfactory and many fatal results. On the other hand, accumulating experience has demonstrated that, although a considerable number of mild and of more severe but quite acute cases of Graves' disease, recover spontaneously or as the result of careful medical treatment, there is a very large percentage of cases that are unrelieved by, or even progress unfavorably in spite of, the best medical care and efforts at environmental readjustment. It is among these that surgeons of acknowledged critique and experience are reporting excellent results and many cures—"cures" not only immediate and symptomatic, but maintained for several years, as determined both clinically and by laboratory tests.

The increasing acceptance of surgical therapy in toxic goiter, in which so many internists share, has come, it would seem, from the more careful selection of cases, which growing experience has brought about. This selection excludes those advanced cases with greatly increased basal metabolism, severe toxic and nervous phenomena and irreparable degeneration in the myocardium and other tissues. (Even these advanced cases, however, are not excluded by Crile as risks too grave for surgical attack). In addition to the simple, including the endemic, type of adolescent goiter, there should also be spared from surgical interference the mild cases of Graves' disease and the early cases, unless and until appropriate non-surgical management fails to control them.

There should not be marked for the operating table every case of thyroid enlargement, nor every case, with or without enlargement, with symptoms that are or appear to be those of hyperthyroidism. While it is true that very often the thyroid basis of nervous manifestations is overlooked, on the other hand the opposite mistake may be easily made of attributing to thyroid intoxication various effort-syndromes and neuroses that have no related somatic disturbance. (The development of Graves' disease from, or after, psychic traumata has, however been repeatedly demonstrated). To suggest to a woman (or man) the necessity for operation merely because of a thyroid enlargement, or a slight prominence of the eyes, or a mild tachycardia and "nervousness", does an immense amount of mischief, as common experience has repeatedly shown; it often originates the very train of symptoms for which the operation is suggested as a cure, and it certainly aggravates those, if any, already present. It is a worse sin than that of planting the need for appendicectomy in the mind of every patient with right-sided abdominal pain or tenderness.

An analysis of the articles here abstracted indicates that while polar ligation and hemi-thyroidectomy are by no means—now, at least—to be abandoned, the best results in toxic goiter are secured by subtotal thyroidectomy. In Porter's very interesting article (*Surgery, Gynecology and Obstetrics,* May, 1923), of which only his conclusions are presented in our abstract, he confesses injury to one or both recurrent laryngeal nerves in ten per cent. of his operations (although most of such injuries were in earlier cases, and in those in which the gland was lifted up from its bed in the performance of a lobectomy). Such a surgical accident should, of course, be avoided (under local anesthesia it is less

likely to happen); but it probably happens more often than is recorded, and even more often than it is recognized. As Porter points out, only by a laryngeal examination can the presence of paralysis of the nerve be excluded.

Of the various tests of thyroid activity, the determination of basal metabolism is recognized as the most important, not only as an aid in the indication for treatment, but also in determining the degree and permanence of "cure". As with so many other tests in medicine, however, it is—so far, at any rate, as concerns the selection of cases for operation—not by itself alone to be allowed to displace clinical judgment.

REACTIONS TO TISSUE DESTRUCTION.

We have given much space in this issue to an article by the well-known experimentalist, Dr. Fenton B. Turck, because it is an important contribution from the side of biology to certain grave surgical problems. It represents a quarter-century of experimental investigations and, while much of the results of these has already been published in various American and foreign journals, this article is their summation to date, it deals especially with the kidney lesions caused by the absorption of tissue toxins (as from burns) and it carries still further the author's studies in producing artificial immunity to such toxins. Turck explains surgical shock, such as is produced by intraabdominal manipulations, not as a reaction through the nervous system, but as the result of the absorption of the products of tissue breakdown. This explanation of the cause of shock appears to have the support not merely of his own precise experimental demonstrations, but also of surgical experiences, notably those acquired during the great war. We commend to our readers the careful study of this article.

Communication

PROGRESS IN SKELETAL EXTENSION.

31 North State Street.
Chicago, Ill., April 17, 1923.

Editor, AMERICAN JOURNAL OF SURGERY:

Dr. A. L. Soresi has done me the honor to comment, in the JOURNAL, February, 1923, upon a statement made by me on the subject of skeletal extension in your number for December, 1922. The statement he quotes from my article is: " The great advance involved in the use of the skeletal extension principle is to be credited to the Swiss, Steinmann, etc."

Dr. Soresi, with the urbanity of manner and expression characteristic of his national origin, points out, without offense, that "the credit for the use of skeleton extension is entirely due to Italy."

Will you kindly let me reply to him that my assertion as he quotes it is exactly correct? I did not say that the "skeletal extension principle is to be credited to Steinmann." I stated only that *the great advance* (i. e., in the treatment of fractures) *involved in the use of the skeletal extension principle is to be credited to Steinmann.* And this is literally and exactly true.

Vanghetti and Codivilla gave us the principle of making extension in fractures by using apparatus percutaneously applied to a limb peripheral to the fractured bone—*not to the injured bone itself.* This is called skeletal extension. *It has not been generally accepted as a practical method of applying the principle of skeletal extension; but Steinmann added a minor, but most practical principle of percutaneous extension applied to the very fractured bone itself,* and this further principle *has been accepted as practically valid throughout the world.* It is used in common practice and, today, gives us that great advance in fracture therapeutics which has come out of the wide, general skeletal extension principle of Vanghetti-Codivilla.

Truly yours,

WELLER VAN HOOK.

Progress in Surgery

Selections from Recent Literature

What Types of Goiter Should Receive Medical Treatment? DONALD GUTHRIE, Sayre, Pa. *The Atlantic Medical Journal,* May, 1923.

The diagnosis of toxic goiter is often missed entirely and that valuable time is lost in treating the patient by medical measures when surgical intervention should have been instituted at an early date; also, often in spite of a correct diagnosis valuable time is lost by employing medical instead of surgical treatment.

Endemic goiter can be prevented by the simple administration of small amounts of iodin twice a year.

The patient ill with early pulmonary tuberculosis, an effort syndrome, a psychoneurotic state, a cardiac disease, or a paroxysmal tachycardia, and who has a simple goiter should be safeguarded from surgical advice by painstaking study. This should include rest in bed under observation, with frequent pulse and temperature chartings, and a careful physical and clinical examination with several basal metabolism tests. Much unnecessary thyroid surgery would be prevented if this plan could be carried out in every case. Occasionally we see well-pronounced exophthalmic goiter in the very young.

Colloid goiters cause no form of intoxication, as a rule are symptomless, and should be treated by medical measures unless pressure symtoms are present or there is unsightly deformity. The large solitary colloid cystic goiter had best be treated by operation. Cancer of the thyroid which can be diagnosed clinically before operation, cannot, in the writer's opinion, be cured by operation. Any goiter which is found to be substernal or intrathoracic upon examination should be operated upon, for delay in these cases may prove hazardous.

We distinguish two types of adenomatous goiter—the non-toxic and the toxic. The non-toxic adenomata appear early in life, but the symptoms of hyperthyroidism do not develop until midlife.

The adenoma remains in a quiescent stage for many years. Treatment for the non-toxic adenoma should be prophylactic, to prevent later thyroid intoxication. Nontoxic adenomata which become cystic or undergo calcerous degeneration should be looked upon as surgical. Iodin and thyroid extract should not be administered to patients with quiescent goiters for fear of changing them into an active state. *X*-ray

and radium are also valueless.

The treatment of the toxic adenomatous goiter should be surgical, and surgery should be employed early, during the insidious onset of the hyperthyroid symptoms, for the recovery after thyroidectomy is prompt and striking and the patient is safeguarded against the myocardial degenerations which often follow prolonged periods of thyrotoxicosis. *X*-ray and radium are valueless in the treatment of the toxic adenomatous goiter.

The treatment of the acutely sick exophthalmic goiter patient, with a basal metabolism of plus sixty or over and with extreme symptoms of the hyperthyroid state, is essentially medical. It is best carried out in an institution accustomed to handling goiter patients and should be psychical as well as medical. Absolute rest, mental and physical, are most important and no operations should be attempted during a rising metabolism or an exaggeration of the symptoms.

The preparation of these acutely sick patients for operation can be greatly facilitated by the use of *x*-ray and radium.

There are undoubtedly milder or abortive types of exophthalmic goiter which return to health with or without treatment. There are also milder continued types of hyperthyroidism with mild symptoms of the hyperthyroid state and with metabolism readings under thirty in whom medical treatment gives varying results.

Frequent metabolism tests are taken after *x*-ray treatments and if the rates and the symptoms remain stationary at the end of eight or ten weeks, operation is advised.

For certain selected mild forms of hyperthyroidism it is safe to give medical treatment a thorough trial, providing the patient is willing to cooperate in every way and will consent to surgical treatment should medical measures fail.

Analysis of My End-Results in Thyroid Surgery. CHARLES A. PORTER, Boston. *Surgery, Gynecology and Obstetrics*, May, 1923.

Concluding from his analysis Porter says: The *x*-rays, through the action on the thyroid and possibly on the thymus, will diminish the risk of operation and will permanently cure certain cases of Graves' disease. If six months' treatment with *x*-rays is not effectual, operation is indicated. The best index of a cure of Graves' disease is a permanent reduction of the basal metabolism to normal. Whether the patient will be cured or not depends on the stage of the disease at which treatment is instituted and the degree of irreparable damage which has already resulted. Though ligation and hemithyroidectomy may cure many patients, ultimate subtotal thyroidectomy most quickly and permanently reduces the metabolism to normal. The psychoses of this disease, which seems frequently to occur in neuresthenic individuals, requires long after-treatment.

The Surgical Treatment of Goiter. HAROLD L. FOSS, Danville, Pa. *The Atlantic Medical Journal*, May, 1923.

Most goiter patients are being menaced by long periods of temporization with all sorts of therapeutic vagaries which in most instances accomplish nothing, but by the delay that goes with many such make-shifts the opportunity is furnished for an insidious and progressive disease to destroy the heart muscle and to demoralize the nervous system.

No medicine, no drug, no therapeutic measure of any sort except surgery should be utilized, except as adjuncts, in the handling of patients with goiter other than of the colloid malignant forms. Radium and the *x*-ray are valuable agencies in the preliminary treatment of very toxic cases, and in certain skilled hands may produce results in the true exophthalmic form of goiter equal to those of surgery, but the process of treatment is long and often tedious and that the likelihood of relapse is great.

The small non-toxic colloid goiters of adolescence are physiologic, usually disappear spontaneously and if treated at all should be treated by the physician.

Innumerable patients have been subjected to needless operations on the diagnosis of exophthalmic goiter when the symptoms were merely those of effort syndrome, disordered action of the heart, neurocirculatory asthenia or functional neurasthenia; but on the other hand even more persons with marked hyperthyroidism have been set down as cases of simple goiter and left untreated merely because the one sign of exophthalmos was absent, while all the other and equally important and thoroughly characteristic evidence of the disease were disregarded.

The results of basal metabolism determinations, blood sugar estimations, the Goetch test, the Kottman reaction, etc., if they are utilized, should merely be placed in evidence with other signs elicited in the general examination of the patient, and of these, the basal metabolism test is by far the most valuable.

Surgery offers a great deal in the treatment of goiter, far more than any other therapeutic means, but that it must be most carefully regulated, the patient thoroughly treated in an institution where the staff is constantly in touch with this type of work, where the internist, surgeon and pathologist have equal opportunities for the study of the disease and where the team work is perfect.

The Roentgen Rays in Thyroid Therapy. MORRIS I. BIERMAN, St. Louis, Mo. *Minnesota Medicine*, May, 1923.

Roentgen-ray therapy is of benefit only in selected cases, which show undoubted metabolic hyperactivity. The adenomatous and the exophthalmic types are especially favorable. The borderline patients are usually the ones in whom we find the least response to treatment. Many of these are really not hyperthyroids at all, but other intoxications with similar manifestations. The others of this group of borderline cases we must admit as failures.

The simple goiter, the colloid, the cystic, and the goiter of adolescence are not proper subjects for roentgen-ray treatment.

Laryngeal Paralysis Associated With the Jugular Foramen Syndrome and Other Syndromes. GORDON B. NEW, Rochester, Minn. *The American Journal of the Medical Sciences*, May, 1923.

New reviews the cases in the literature and adds 7 cases of laryngeal paralysis associated with a syndrome due to involvement of the last four cranial nerves and the cervical sympathetic nerve in the region of the jugular foramen (Jackson's syndrome.) When this occurs it is usually owing to a neoplasm, although a tuberculous process in the glands of the neck or an acute phlegmon may be the cause. During the World War cases were reported due to bullet or shrapnel wounds. The vocal cord is in the intermediate or cadaveric position in vagus involvement.

Lung Abscess. GEORGE J. HEUER, Cincinnati. *Minnesota Medicine*, May, 1923.

Heuer discusses the pathogenesis, pathology, diagnosis and methods of treatment of lung abscess and gives this summary of personal experience:

1. Of 62 cases of lung abscess in Johns Hopkins Hospital surgical records 14 were unrecognized during life. Autopsies in these cases showed in every instance scattered small lung abscesses, which could not have been satisfactorily treated by surgery.

2. In the remaining 48 cases lung abscesses were recognized. In these the abscess or abscesses followed pneumonia in 27 cases; they were complications following some surgical operation in 11 cases; followed acute abdominal infections in 5 cases; and were complications following a miscellaneous group of conditions in 5 cases. The abscesses were associated with empyema in 12 cases; not so associated in 36 cases.

3. The treatment of these 48 cases consisted of:

(a) Drainage in one or two stages in 36 cases.
(b) Drainage followed by lobectomy in 2 cases.
(c) Exploratory thoracotomy followed by collapse therapy in 5 cases.
(d) Postural treatment in 3 cases.
(e) No treatment in 2 cases.

Forty-three patients therefore were subjected to 45 operations. Following operation there were 13 deaths, an operative mortality of 28.8%. Eleven deaths followed drainage operations and autopsies showed:

(a) In 6 cases there were multiple abscesses, only one of which had been drained.
(b) In 1 case there was a consolidation with softening and abscess formation involving the entire lung.
(c) In 1 case there was an acute spreading gangrene involving the entire lung.
(d) In 1 case there was an extensive broncho-pneumonia

in addition to the abscess.

(e) In 2 cases there was an extensive inoperable carcinoma of the esophagus with extension into and with abscess formation of the lung.

Two deaths followed lobectomy performed some months after a drainage operation. Autopsies showed that one death was due to an extensive broncho-penumonia and the other to a pleural infection.

The end-results in the 35 patients who recovered at the present time are:

(a) Of the 25 cases that recovered following drainage operations 16 are well from 1 to 10 years, 5 are entirely well, 4 have not been heard from.

(b) Of the 5 cases that recovered following exploratory thoracotomy plus collapse by artificial pneumothorax, 2 are well from 1 to 10 years, 3 have not yet been heard from.

(c) Of the 3 cases treated by postural methods, 1 is not entirely well, 2 have not been heard from.

(d) Of the two cases who refused operation or other treatment neither has been heard from.

The Etiology and Clinical Features of Lung Abscess. JOHN HOMANS, Boston. *The Boston Medical and Surgical Journal*, April 19, 1923.

That lung abscesses, due to all causes, have a strong right-sided and lower lobe preponderance is attested by all available statistics. This series gives: Right—Upper, 4; middle, 3, lower, 7. Left—Upper, 4; lower, 5. Total right-sided, 14, or 61%; total left-sided, 9, or 39%; total lower, 12; total upper, 11; and by taking Lockwood's chart of accurately located abscesses from all sources, much of the same findings are obtained.

The distribution of lung abscess shows a proportion of right lung infections very similar to that found in lobar pneumonia, and the right lower lobe is involved quite as often in pneumonia as in abscess. The figures given by Rupp for the localization of embolism and infarction in a large series of postmortems shows a preponderance of right-sided lesions almost identical with that of abscess.

The problem of treating lung abscess becomes very much simplified if we admit, as a study of etiology has suggested, that post-operative abscess is a less serious disease than that which arises from other causes. The point has been made by many other writers.

In this series of 23 lung abscesses 13 were post-operative. Of these post-operative abscesses, seven recovered with or without operation and five were not cured. Of the abscesses which arose from pneumonia and various other causes, three recovered and six were not cured.

Treatment of post-operative abscess: Of the 12 patients in whom the results of treatment of this type of abscess are known, two were not operated upon and both recovered. Non-operative treatment, advised by the surgeon or insisted on by the patient in two other instances, resulted in such deterioration of the patient that one died of the disease several years after operation, and the cure of the other was much delayed. Among the successful operative cases, there was delay of six months in one, four and one-half months in another, and three months in a third. Indeed, a delay is often quite clearly of advantage in the case of a very sick toxic patient, who in the acute stage would evidently succumb to any operative procedure.

Every patient seen within the first few weeks of the establishment of an abscess should be treated by postural drainage, the best food, hygienic surroundings and antiseptic inhalations; this treatment should be pursued so long as the general condition continues to improve, and so long as the signs in the lung, as shown by physical examination and *x*-ray study, are retrogressing; but the patient should be under close observation to detect a change for the worse or cessation of progress. Operation is likely to be fatal in very sick patients. If conservative treatment will not tide a patient over the acute stage of the disease, operation is not likely to do so. It is highly probable that when an abscess becomes chronic under conservative treatment operative drainage is the only measure likely to prove curative.

The treatment of lung abscess of other than post-operative origin: (Abscesses originating in pneumonia, from inhalation of poisonous or irritating substances and those in which no etiologic factor is known.) These cases are far more dangerous than the post-operative variety and they have proved more difficult of cure. Wherever gangrene of any consequence has been associated the result has been fatal, and operation has evidently hastened the death.

The *x*-ray shows fewer clean-cut abscesses and more diffuse clouding of the lung than with abscesses of the post-operative type.

Operative technic: Stereoscopic *x*-ray plates and physical signs permit accurate localization. The ease with which ribs can be resected under a local anesthetic takes the operator to the pleura over the abscess with little disturbance of the patient. Owing to the frequency with which the lesion is found close to the surface of the lung, the pleura is usually adherent. Thus, the lung can be explored for abscess with little danger of pleural infection. Puncture of the chest wall with a sharp needle never should be used. When the pleura is exposed and the lung found adherent, the abscess is most easily found (if its location is not at once apparent) by passing into the lung before a fluoroscope a blunt needle, such as for ventricular puncture. Or if the fluoroscope is not available, a short blunt needle or wire may be passed in at a guess and held in place by packing while stereoscopic *x*-ray plates are made. Even a small abscess can easily be found in this way, and when found may be entered by a blunt instrument, exploited with the finger and drained with a soft tube. The rare deep-seated abscesses are not so hard to find as they are difficult to drain, for if the lung about them is considerably scarred, it is difficult to enter except with a knife, a procedure which must be considered, as regards hemorrhage, a pure gamble. Once the abscess has been entered, cauterization of the tract leading to it stops bleeding and keeps the tissues from healing too soon.

When the lung is not adherent, operators differ as to the proper course to follow. The procedure of fearlessly making a pneumothorax and examining the lung with the fingers is not perhaps objectionable in the milder and more chronic cases, but where the balance between life and death is fairly even, such disturbance of the pus-soaked and partly gangrenous tissue is likely to spread infection.

The procedure to be used when the lung and pleura are not adherent will depend upon the patient's condition and the relations of the abscess. Artificial pneumothorax has a definite place here. In the treatment of the general run of abscesses pleural adhesion must render an attempt to collapse the lung more or less ineffectual, but where a free pleural cavity is demonstrated pneumothorax deserves more of a trial.

Some Observations on the Treatment of Acute Appendicitis. R. J. MCNEILL LOVE, London. *The British Journal of Surgery*, April, 1923.

In cases of appendicitis requiring drainage of the pelvis or the iliac fossa, Love recommends that this should be through a stab wound suprapubic or in the flank respectively, rather than through the operative incision. This favors primary union of the original wound, makes adhesions and fecal fistula less likely to develop and avoids the risk of secondary hemorrhage from erosion of the external iliac vessels. He also says:

All cases of acute appendicitis should be operated on immediately if a band of hyperaesthesia is still present.

Under appropriate treatment the majority of remaining cases subside, and the appendix may be removed seven to ten days after the temperature and pulse become normal. As compared with emergency operation this line of treatment shows a lower mortality, fewer complications, and a shorter stay in hospital.

In cases in which expectant treatment fails, twenty-four hours' delay does not appear to influence the prognosis adversely.

Except in cases where difficulty is anticipated (e. g., recurrent cases), or where the diagnosis is doubtful, the gridiron is the incision of choice, as manipulations can then be limited to the infected area. In other cases a right rectus incision, displacing the muscle outwards, is preferable to Battle's, there being less risk of injury to the nerves.

The Chronic Abdomen. ROBERT HUTCHISON, London. *British Medical Journal*, April 21, 1923.

The symptoms of the "chronic abdomen" are many, various, and ever-renewed. Some refer directly to the abdominal organs, others are more remote and general, but they are always described with great prolixity and detail. Amongst those most commonly complained of are abdominal aches and pains of various sorts and places, but especially in the right iliac fossa. Instead of pain the patient may speak of a "raw feeling inside," or of "an indescribable sensation in the stomach," or of a "dragging." Constipation almost always figures prominently in the list of symptoms, and flatulence is also frequent. In the treatment of the chronic abdomen the most important thing is to catch the patient early. If she has once set her feet on the slippery slope which leads to successive operations she is undone. A timely "fattening cure," followed by the provision of an efficient abdominal support, regulation of the bowels by the mildest laxative, supplemental if necessary by simple enemas, an abundant but unirritating diet, soothing of the nervous system by bromides—these are the physical measures. On the mental side, the patient should be encouraged to take up some definite occupation which will provide her with an interest outside herself.

The Significance of Diarrhea Following Abdominal Operations. EDWIN BEER, New York. *Annals of Surgery*, May, 1923.

Diarrhea may follow any operation on the intraperitoneal or extraperitoneal abdominal organs. Occasionally this diarrhea is uncontrollable and the cause of a fatal outcome. It may appear out of a clear sky during an uneventful convalescence, either early or late in this period. The exact causation of the severe diarrhea is very difficult to determine and each case should be studied individually. The milder transient diarrheas are probably dyspeptic or irritative in origin. Localized pericolic suppurative peritonitis, ileocolitis and proctitis, perhaps as a terminal infection in weakened patients, or in patients with renal insufficiency, must be considered in looking for an explanation, while it is just possible that too large a stoma may favor the development of this startling complication. Mild cases seem to recover on a simple diet and the usual astringent therapy. Severe cases have baffled all therapy, though if we may reason from analogy, one would expect to control some few of these if due to pericolonic inflammation by a drainage operation. Those in which the complication seems to be of the type of a terminal infection, superimposed on a cachexia or renal insufficiency will probably continue to be rebellious to therapy.

Vascular Occlusion of the Mesenteric Vessels. J. W. TANKERSLEY, Greensboro, N. C. *Virginia Medical Monthly*, April, 1923.

In the symptoms there is nothing pathognomonic. It occurs most frequently in men and about the middle decade of life. Cardiac disease, arteriosclerosis and recent operations are predisposing factors. Acute injury is always a factor to be considered. Pain is very severe and colicky, usually generalized in the abdomen, though it may become local later. If peritonitis sets in, it may again become general or local. When vomiting does occur, it is first bile and the contents of the stomach, and later may become fecal. Obstipation is the rule and, when bowels are moved by means of an enema, frequently small amounts of blood may be found in the stools. This is not indicative but suggestive. Abdomen and muscle rigidity are always present and frequently may be localized over the involved area or mid-abdomen. Distention gradually develops and occasionally a small mass may be felt in the abdomen. The temperature is no guide; it usually falls at first and then gradually rises as peritonitis develops. This is true also of the leucocyte count. Shock is considerable and seems to be greatest with the onset.

The pathology is most often a thrombosis in some part of the superior mesenteric vessels and the mucosa of the intestines suffers first and most. This gives the blood in the stools.

Occasionally an embolus or thrombus may cause no apparent symptoms; or, if symptoms do occur, they may clear up, due to the establishment of collateral circulation. This is accounted for possibly by the slowness in closure of the vessel. The permanency of the collateral circulation is to be doubted as cases have been reported where it broke down subsequently and caused infarction with death.

Intestinal obstruction may occur without infarction, as the blood supply to the parts may be sufficient for nourishment, but not for function.

Should infarction occur, as shown by the degree and progress of the symptoms, excision of the infarcted segment, offers the only solution.

On Acute Purulent Processes in the Intestinal Wall, a Contribution to the Knowledge of Phlegmonous Enteritis. GÖSTA BOHMANSSON, Sweden. *Acta Chirurgica Scandinavica*, April 11, 1923.

Limited purulent processes localized to the intestinal wall, not proceeding from the appendix or preformed diverticulum, are not so rare as would seem from the literature on so-called phlegmonous enteritis, although the actual picture of a diffuse phlegmon of the intestinal wall is not always established in these cases. The difference between these phlegmons and other purulent affections of the intestinal wall is only gradual. The disease can appear in any part of the intestinal canal, and seems to be most usual in the colon. It is most often of enteral origin, although a hematogenous modus in certain cases cannot be denied. The course varies considerably in different cases with oscillations between a violently acute course and transitory forms of the chronic inflammatory tumors. The process can heal spontaneously at any stage without leaving any after-trace, or with fibrous cicatrization and strictures in the lumen.

In several cases a mechanical insult has been confirmed, which may be presumed to have served as port of entry for the infection. Different bacteria have been verified as causal agents for the morbid process. In the more acutely progressing cases it is commonly a streptococcus. A clinical diagnosis is hitherto never made before the operation and the symptoms vary considerably according to the different localization and the different degree of virulence of the casual bacteria. Operation is always indicated if the patient's condition allows of any interference. Although several cases of spontaneous healing are known, yet the result from resections performed speak in favor of this method of treatment being followed when technically possible.

Chronic Duodenal Ileus. SEYMOUR BARLING, Birmingham. *The British Journal of Surgery*, April, 1923.

The condition of duodenal ileus may arise from pressure of the mesenteric root containing the superior mesenteric artery and vein on the third part of the duodenum.

Whilst probably such obstruction is the chief predisposing cause of acute dilatation of the stomach, it may also give rise to chronic digestive disturbance, simulating chronic ulcer of the stomach and duodenum, or inflammation of the appendix or gall-bladder, and is sufficiently common to be recognized as a clinical entity.

Apart from the direct effects produced by obstruction, duodenal ileus plays a part in the etiology of some cases of chronic gastric and duodenal ulceration and infections of the biliary tract.

In many cases an accurate pre-operative diagnosis of the condition may be made by a careful consideration of the clinical symptoms and the results of a radioscope examination.

When performing laparotomy the condition must be borne in mind and sought for as a cause of chronic indigestion, and, in the presence of hypertrophy and dilatation of the stomach and duodenum with patent pylorus, the operation of duodenojejunostomy performed.

Roentgenologic Signs of Cancer of the Colon. RUSSELL D. CARMAN, Rochester, Minn. *The Journal of Radiology*, May, 1923.

More than 90 per cent of colonic cancers will show roentgenologic evidences of a lesion when examined with the opaque enema.

A diagnosis of cancer cannot be made on the roentgenologic findings alone, for the condition is stimulated by various lesions, and especially per peridiverticulitis.

Cancers of the cecum are more likely to elude observation by the roentgen ray than those situated elsewhere in the colon.

Ring cancers are easiest to find with the roentgen ray.

The exclusion value of a negative finding should neither

be overestimated nor underestimated. In some cases the roentgen ray will fail to disclose a lesion, but not more than one in ten cancers should escape detection.

Roentgenologic examination of the colon in cases of cancer will not throw light on the question of operability, since the growth may be resectable regardless of its location, extent, or the presence of adhesions.

A Note on the Operation for the Radical Cure of Femoral Hernia. ERNEST W. HEY GROVES, Bristol. *The British Journal of Surgery*, April, 1923.

The hernia is exposed by a vertical incision about four inches long, the middle of which corresponds with the fold of the groin. A very little dissection then reveals the hernial sac, lying on the pectineus muscle, with Poupart's ligament crossing its neck, and on its outer side the femoral vein joined by the long saphenous vein. The attachment of Poupart's ligament to the pubic spine is then cut close to the bone and the ligament turned outwards. This gives a clear view of the neck of the sac, which is then opened and its contents dealt with according to their condition. Should resection of the bowel be necessary, this can be done perfectly well by the exposure thus afforded. When the sac has been cut off and its neck ligatured, the femoral ring and canal can be obliterated. The conjoined tendon is defined and sewn down to Cooper's ligament and the upper edge of the pectinal fascia. The outer one or two stitches also take up the femoral sheath on the inner side of the femoral vein, so as completely to close the abdominal cavity from the thigh. Poupart's ligament is then laid over the line of suture between the conjoined tendon and Cooper's ligament, beginning from the vein and working inwards towards the spine of the pubis. As the inner attachment of Poupart's ligament has been divided, there is no tension in this structure and it can be made to follow the line of the pubic bone, and the final attachment of its cut end will be about one-half inch further outwards than it was originally.

Advantages of this combined femoral and inguinal operation are: 1. In cases of strangulated hernia it gives ample room to deal with damaged bowel and, if necessary, to do a resection. 2. It gives all the advantages of the inguinal approach, i. e., the possibility of closing the femoral canal from above, without the necessity of trying to drag up the hernial sac through the femoral canal.

Ulcer of the Stomach and Duodenum. E. STARR JUDD, Rochester, Minn. *Minnesota Medicine*, May, 1923.

The change seen in the roentgenograms a few days or weeks after institution of medical treatment is not due to healing of the ulcer. The ulcer has always been present in the several cases in which Judd operated under such circumstances.

The surgical management of an ulcer of the stomach should always include removal of the ulcer. If the ulcer is small, it should be excised and a gastro-enterostomy performed. It is necessary to perform a gastro-enterostomy in order to maintain proper peristalsis in the stomach. Complete inability of the stomach to empty may result from excision of an ulcer from the lesser curvature, even when the ulcer is at some distance from the pylorus. If the ulcer is large, and especially if it has a posterior attachment, as is often the case, it is best to perform a sleeve-resection, taking a part of the greater curvature, even though it is not involved. This will maintain normal peristalsis and gastro-enterostomy will not be necessary. If the ulcer is near the pylorus it is often best to perform a pylorectomy, to complete the anastomosis by end-to-end suture, or by closing the ends, and gastro-enterostomy. A gastro-enterostomy alone in these cases leaves the possibility of malignancy, and, furthermore, one-third of the patients will not be relieved by gastro-enterostomy.

The surgical treatment of duodenal ulcer is somewhat different from that of gastric ulcer. It is not necessary to consider the possibility of present or future malignancy. If the ulcer is in the free portion of the duodenum so that a technically accurate excision can be made, the operation of choice is complete removal of the ulcer and restoration of the duodenum. This is a simple and safe procedure. In excising a duodenal ulcer it is not necessary to perform a gastro-enterostomy. If the stomach is large with a tendency to spasm, then not only is the ulcer excised, but the sphincter at the pylorus is divided, in order to enlarge the pylorus and overcome the spasm. If the duodenum is not free and cannot be mobilized so that the ulcer may be accurately excised, a gastro-enterostomy alone should be performed.

The Clinical Importance of the Chronic Changes in the Appendix Which Are Discovered by the Roentgen Ray. FRANKLIN W. WHITE, Boston. *The Boston Medical and Surgical Journal*, April 19, 1923.

The *x*-ray examination of the appendix discovers many things about the appendix which were unsuspected, and incidentally many things which may not trouble the patient much, such as poor mobility, small fecal masses, peculiar shapes, chronic obliteration of lumen, "beading," moderate delay in emptying. These were common signs in cases, which were not operated, going about for one to four years after the *x*-ray examination with practically no digestive symptoms whatever. The moral is that when these signs are discovered by the *x*-ray we must not make too much of them, consciously or unconsciously. Many persons will be helped by having a "chronic appendix" removed, many others will get no help at all.

"Chronic appendicitis" is not an entity. The changes vary greatly in degree and kind and in what should be done about them, all the way from the mild, harmless chronic obliterative type to the kinked, bulbous, tender, badly draining type. The atrophic obliterated organ of middle life and beyond usually gives no symptoms and makes no important trouble.

There is no organ so often removed without cause merely because of some anatomical change.

A Suggested Method for Protecting the Ovaries from Post-Operative Radiation. (*Vorschläge für den Schutz der Ovarien bei postoperativer Nachbestrahlung.*) WALTER OFFERMANN, *Zentralblatt für Gynäkologie*, April 21, 1923.

After removal of the uterus for malignant tumor, the practice of post-operative radiation of the pelvis has become universal. In so doing the ovaries, if left behind, are sure to be affected and rendered inert by the rays, thus adding menopause symptoms to the patient's troubles.

Offerman suggested that at operation the ovaries be removed, cut into slabs of from 1-2 mm. thickness and implanted in the rectus abdominis, above the umbilicus. In this situation the transplants can readily be sheltered from the rays.

Treatment of Beginning Incarceration of the Pregnant Retroflexed Uterus. (*Zur Behandlung der beginnenden Inkarzeration des retroflektierten, graviden Uterus*). F. UNTERBERGER. *Zentralblatt für Gynäkologie*, May, 1923, 47; 714.

The usual methods have consisted in attempts at manual reposition in the lithotomy or the knee-chest position first without, and if this is unsuccessful, then with anesthesia. Traction by means of a volsellum applied to the cervix has been tried, and finally reduction by a colpeurynter filled with mercury has been advised.

Unterberger has used the following simple method, which does not jeopardize the existing pregnancy, with success in three cases, in one of which ischuria paradoxa and bloody urine had already appeared.

The patient is placed in very steep Trendelenburg posture after the bladder has been emptied with a soft catheter. If the uterus does not rise into the abdomen in ten to fifteen minutes, the maneuver is repeated next day. In all three cases the pregnancies continued to term.

A Critical Analysis of the Kjelland Forceps based on 54 Personal Cases and 1,000 from the Literature. (*Zur Kritik der Kjellandzange, an Hand von 54 eigenen Beobachtungen und etwa 1000 mitgeteilten Fällen der Literatur.*) KARL FINK. *Zentralblatt für Gynäkologie*, April 28, 1923.

Fink subjects the question of the superiority of the Kjelland forceps to a careful review. The especial advantages claimed by the inventor are easy application in whatever position the saggital suture may lie at any level of the pelvis, and absence of any tendency for the forceps to slip off.

The head is also said to rotate within the forceps without endangering the child.

Both from personal observation, which includes two maternal deaths due to uterine perforation, and from the literature, Fink concludes that the new instrument is rarely superior to the classic models and often proves a dangerous tool even in skilled hands.

Pregnancy Complicated by Cancer of the Cervix (*Über die Komplikation der Schwangerschaft mit Gebärmutterkrebs*). BERNHARD SCHWEITZER. *Zentralblatt für Gynäkologie*, April 28, 1923.

Eleven cancer cases were found among 23,500 pregnancies, about 0.05%, which corresponds to current statistics.

25-30 years 1 case.
31-35 years 5 cases.
36-40 years 2 cases.
41-45 years 3 cases.

Average age 35.5 years. Plurigravidae predominated, three being in their seventh pregnancy. In three cases the bleeding had begun before pregnancy had developed. In one case the family physician mistook the cancer for placenta previa.

In two cases abortion supervened, one dying of sepsis.

Pregnancy appears to increase the growth rapidly of cervical cancer, but does not aggravate its malignancy. Of 11 cases, 82% were operable, and of these 43% were cured. The earlier in pregnancy operation is resorted to the better the outcome. Radical removal, if necessary preceded by Cesarean section, is the method of choice. Post-operative radiation is indicated.

Management of the Female Urinary Bladder After Operation and During Pregnancy: A Further Study of the Residual Urine in Its Bearing on Urinary Tract Disturbances. ARTHUR H. CURTIS, Chicago. *The Journal of the A. M. A.*, April 21, 1923.

This is a study devoted to post-operative care of the urinary bladder, with especial consideration of the part played by residual urine throughout the period of convalescence of 1,595 female patients subjected to serious operations. Use of the catheter has been avoided when possible, but no patient has been allowed to suffer from distention. Residual urine has been present after the return of spontaneous micturition in more than 64 per cent. of all repeatedly catheterized patients. The most important principle in the treatment of such cases has been the daily passage of a catheter, immediately after urination, until residual urine is no longer present. Only three of those who were catheterized daily until the return of normal function developed urinary tract infection. These three subsequently showed residual urine after they were thought to have returned to normal, and it is believed that lack of treatment during this time accounts for the infection. Laboratory study of urine from patients subjected to post-operative catheterization reveals that there may be temporary cystitis. This invariably—and usually very promptly—disappears if freedom from residual urine is maintained. Instillation of ⅛ per cent. silver nitrate and administration of urinary antiseptics by mouth are helpful adjuncts in the treatment. Strict avoidance of the use of the catheter after all operations despite distress, would perhaps reveal equal freedom from infection; but this plan cannot be followed without much suffering to many patients. Moreover, animal experimentation indicates that if bladders are allowed to distend and overflow, considerable back-pressure destruction of kidney tissue results. Clinical evidence indicates that residual urine occurs very often in pregnant women. The notable frequency with which it is present in those with pyelitis of pregnancy suggests that ascending infection from this source is of considerable importance.

Fracture of the Head and Neck of the Radius. JACOB GROSSMAN, New York. *New York Medical Journal and Medical Record*, April 18, 1923.

Fracture of the head and neck of the radius is not an uncommon condition; 16 cases having been present in a series of 150 cases of elbow fractures. It may result from a fairly mild trauma, direct or indirect.

The best position and method of treatment is that of acute flexion. Baking should be instituted from the first. Motion, either active or passive, should be delayed until the fluid has disappeared from the radiohumeral joint.

In arriving at a diagnosis one should always eliminate the presence of so-called traumatic palsy. The prognosis is good in all cases except in those in which the fragments are so small and displacement so marked that they cannot be replaced. In these cases operative interference must be resorted to.

Actinomycosis of the Right Iliac Fossa. R. ST. LEGER BRICKMAN, Sheffield. *The British Journal of Surgery*, April, 1923.

Actinomycosis in this situation is more common than is generally supposed. Cases of infection of the cecum have been reported, but no recorded case of a similar condition in the walls of the appendix can be found. The appendix plays purely a secondary part in the etiology of this disease of the right iliac fossa. It acts as a locus minoris resistentiae through which the organism escapes into the retroperitoneal tissues. Conservative surgery gives the least unpromising results. It should be limited to simple incision, evacuation, and drainage. Resection and extensive curettage should play no part in treatment. Large doses of potassium iodide, together with an early administration of autogenous vaccines, offer the best hope of cure.

The Treatment of Internal Derangements of the Knee-Joint. A New Method of Operative Exposure. A. G. TIMBRELL FISHER, London. *The Lancet*, May 12, 1923.

Transfixion and local removal of a loose body is an unscientific relic of preantiseptic days. A full exposure of the joint is imperative.

Curved or straight lateral incisions over the joint interval, save in very exceptional circumstances, do not yield such satisfactory results as complete exposure, and through them it is impossible to explore fully the anterior compartment of the joint save by digital examination. The finger under no circumstances should be introduced into the joint.

The transpatellar route undoubtedly gives a good exposure of practically the whole of the anterior compartment. The method described is simpler in performance, gives a better exposure, is a less severe operation, since iit involves no division of bone or articular cartilage, is less likely to be followed by osteo-arthritic changes, and is attended with a shorter convalescence. It appears to have been hitherto overlooked that there are objections on physiological grounds to the transpatellar route, particularly if the most accurate apposition of bone and cartilage be not obtained. It has been shown that no true cartilaginous repair takes place in incisions in the center of articular cartilage, but that incisions, if they heal at all, do so by an imperfect fibrous tissue. It appears that in these cases, particularly when apposition is not perfect, the irregularity and imperfect union of articular cartilage actually act as a source of irritation and may, particularly in individuals past the meridian of life, set up a form of traumatic osteo-arthritis.

The author's operation: The stages described are particularly applicable to cases where symptoms point to the inner side of the joint. If symptoms point to the outer side of the joint, the positions of the incision in the skin and capsule are reversed.

Stage 1: A slightly curved incision through the skin and subcutaneous tissues commences in the midline an inch above the uppermost limits of the suprapatellar pouch, skirts the inner border of the patella and the inner border of the ligamentum patellae, and ends below slightly to the inner side of the tubercle of the tibia. The flap of skin and subcutaneous tissue is reflected outwards to slightly beyond the outer border of the patella. The skin flaps are protected by gauze, or the incision may be made through a covering of sterile gauze or stockinette which is afterwards clipped to the skin edges.

Stage 2: Make a midline incision through the well-defined layer of fascia covering the quadriceps tendon. Prolong this downwards and slightly inwards to the middle of the upper border of the patella. Thence continue in the

midline of the patella through the same layer of fascia and through the somewhat thinner fascia covering the center of the ligamentum patellae as far as the tubercle of the tibia. This incision should avoid the subjacent quadriceps tendon, the periosteum on the front of the patella, and the ligamentum patellae.

Stage 3: The above-mentioned fascia is raised and reflected inwards by a few light touches of the scalpel. Above, a few fibers of the vastus internus may with advantage be included in the base of the flap. Below, the base of the flap should be half an inch from the inner border of the patella and from the inner border of the ligamentum patellae.

Stage 4: The fascial flap is to be well retracted inwards. Next the capsule is divided a quarter of an inch from and parallel with the inner border of the patella. The incision is carried upwards somewhat obliquely through the inner fibers of the quadriceps tendon, carefully avoiding any muscular fibers of the vastus internus; and below, the incision is carried through the capsule at the inner border of the ligamentum patallae. The synovial membrane is next divided along the line of division of the capsule save in the extreme lower part of the incision.

Stage 5: The patella is dislocated to the outer side of the joint, and on further flexion an excellent exposure is obtained of the whole of the anterior compartment. With suitable retraction every part is brought into sight.

On completion of the intraarticular stage, the knee is extended and the patella is easily replaced. Suture the cynovial membrane with a continuous suture of fine catgut. The incision in the alar pad should be carefully sutured, but if the latter is pathologically enlarged, it should be removed. The capsule is next sutured with a continuous suture of stout chromicised catgut. Next the flap of fascia is replaced and sutured in the midline with a continuous suture of medium-sized catgut. Thus the opening into the joint is closed after the fashion of a double-breasted coat. The skin is sutured with interrupted sutures. The tourniquet is not removed until dressings and bandages are firmly in position.

No splint is used. The limb is supported in a position of slight flexion with pillows. The skin stitches are removed on the seventh day, and massage and movements, both active and passive, instituted, the patient being allowed to sit in a chair daily. At the end of two weeks the patient is allowed to commence to walk about. Massage and movements are continued. At the end of three weeks patients are usually able to walk fairly well, and at the end of the fourth week almost normally.

Amputations in the Region of the Knee Joint. HOWARD H. DIGNAN. *California State Medical Journal of Medicine,* April, 1923.

The essential points of a good thigh amputation are:

1. Shape. The stump must be cone-shaped, so that the bucket will support part of the body weight on the sided.

2. End-bearing. This is not necessary, but it is a very decided advantage, and is easily obtainable by the Gritti-Stokes method.

3. Scar. The scar should be small, and must not come on the end-bearing surface. It must not be adherent to the bone or it is liable to tear from the skin tension. The scar of the Gritti-Stokes is posterior and protected from pressure by the tendons of the biceps femoris and the semitendinosus.

4. Bone. The end of the bone must be smooth and rounded. It must be free from exostoses and bone spicules. These can be prevented if the periosteum is cleanly cut, no strips being left, and all infection avoided.

5. Nerves. The nerves should be cut as high up as possible, the medullary sheaths ligated over the cut ends, so that nerve bulbs will be avoided and the nerve axones will not grow out into the surrounding scar tissue, muscles, periosteum, and skin to give distressing and painful symptoms.

The Gritti-Stokes operation is the only one that answers all these qualifications. Its superiority has been strikingly demonstrated. Many of these amputes have better function than the below-knee amputations.

After the advent of asepsis, the method was not put to the test until the last war, from which it emerges alone as the suitable amputation for this region. This operation should never be done where there is any chance of infection. It is better to save as much tissue as possible, and then, when safe, a secondary operation can be done. Tuffier states that in his war experience 90 per cent of amputations had to have a secondary operation.

Amputation cases should never be allowed to develop a crutch habit. A week or two only with crutches should be allowed during the time they are learning to walk on a pylon.

The technic of the amputation and of making a pylon are described.

Traumatic Valgus With Dislocation in Lisfranc's Joint. ARCH F. O'DONOGHUE, Sioux City. *The Journal of the Iowa State Medical Society,* April 15, 1923.

Traumatic flat foot is a true dislocation and should be treated as such. A convex inner border of the foot with a prominent cuneiform and fifth metatarsal is the most important physical sign of dislocation in Lisfranc's joint. Operative reduction is usually indicated in chronic cases. Recent and operative cases carry a good prognosis.

Operative Treatment of Certain Fractures of Long Bones. JOHN J. MOORHEAD, New York. *The Journal of the A. M. A.,* April 28, 1923.

The principle of "notching" has been employed in a series of cases and reported, and success by this procedure has been so gratifying that in future many recent and old cases previously subjected to non-operative reduction will be managed by this method. The time to operate is within the first thirty-six hours, if possible, the earlier the better, if there are none of the usual operative contraindications. The fragments are notched by a rongeur, chisel, saw or "bone notcher." Advantages claimed for this method are: (a) More accurate coaptation means firmer, earlier union in more exact alignment. (b) With the assurance that definite coaptation has been obtained, there is less danger of interposition of soft or hard parts which would prevent or impede union. (c) Primary neural or vascular damage is more readily discovered and corrected; secondary neural or vascular damage from pressure or callus inclusion is very unlikely. Disadvantages are: (a) Fractures are put into the operative class with the attendant risks of anesthesia and infection. (b) The method is needlessly severe and dangerous, since good results have heretofore been obtained by simpler methods. (c) It is inapplicable for general use. (d) It produces shortening. This is usually slight and of no importance. The method is particularly adapted to recent displaced or old malunited fractures of the shaft of the radius, ulna, tibia and humerus, in the order named. The femur is not included because skeletal traction is here so effective. Joint fractures are usually amenable to closed correction, perhaps excepting those of the elbow and some at the shoulder and ankle.

The Value of the Roentgen Ray in the Diagnosis and Prognosis of Sarcoma of the Long Bones. HENRY W. MEYERDING, Rochester, Minn. *The Journal of Bone and Joint Surgery,* April, 1923.

The roentgen ray is a most valuable aid in the diagnosis and prognosis of sarcoma of the long bones. It reveals the location, size and extent, and to a certain degree, the structure and origin of the tumor. It is the earliest aid in determining the presence of pulmonary metastasis, although the absence of demonstrable lesions does not mean that metastasis has not occurred. Roentgenograms of the lungs should be made as a routine measure in every case of suspected malignant tumor of the long bones, particularly if removal of the primary tumor is contemplated.

Various types of sarcoma, central or periosteal, with marked differences in density, structure, and location may be observed. The roentgenographic appearance of these types, while not always characteristic. is fairly characteristic, and yields a high percentage of accurate diagnoses. Operation should seldom be resorted to, and amputation never, without exploratory operation with the use of the tourniquet, and microscopic proof of malignancy. There may be exceptions when the member or joint is already so far involved that it is obvious that the extermity cannot be saved.

Meyerding reports 9 cases.

The Results of Staphylococcus Infection of the Bone. WALTON MARTIN, New York. *The Surgical Clinics of North America*, April, 1923.

In connection with other cases Martin demonstrated that of a boy operated upon by him two and a half years ago for acute osteomyelitis of the lower end of the left tibia. He then had a little pain in the right knee and several months later developed a swelling over the upper end of the right tibia. This slowly increased in size, was slightly tender, but not red, and was not associated with fever or leucocytosis. Roentgenograms showed the shadow of the swelling of the soft parts, and also that of a small detached fragment of the bone about 2 cm. from the medial condyle of the tibia, but no change in the tibia itself. The condition was one that might have been—as such cases sometimes are—mistaken for a sarcoma. Incision, under nitrous oxide narcosis, through the expansion of the sartorius tendon disclosed the sequestrum lying in a mass of soft granulation tissue. Neither this nor the underlying bone was curetted or otherwise disturbed. The sequestrum was removed and the wound was closed by sutures, after making a culture from the granulations. This showed staphylococcus aureus. Martin presented the case as a subperiosteal cortical bone infection, metastatic from an osteomyelitis in the opposite leg, demonstrating the attenuation of the virulence of staphylococci in bone infections.

The Use of the Delayed Flap in Secondary Operations on the Palate and Antrum. GORDON B. NEW, Rochester, Minn. *Minnesota Medicine*, April, 1923.

The method which New found most successful in the closure of both the large and the small openings is the application of cleft palate surgery of the principles employed in using the delayed flap in plastic surgery of the face and neck. In bringing down a flap from the forehead to fill in an opening in the nose, or in using a flap from the back to replace scarring of the anterior part of the neck, it is best to raise the flap and then suture it directly back in place, leaving it for a week or ten days before transferring it to the area where it is to be employed. In this manner, the blood supply of the flap is insured and the flap also thickens.

The same principles are applied to the closure of difficult postoperative openings in the palate. The flap is outlined on one side of the opening with the pedicle posterior, and then elevated and placed back in its original bed. At the same time, the opposite side of the opening is elevated from its mesial margin and allowed to fall back. A suture or two may be employed to hold the anterior extremity of the flap in the correct position. In a week's time the flap is again elevated, the mesial margin trimmed and sutured across the opening to the freshened and elevated margin of the opposite side. During the week of the delay the blood supply to the flap is improved a great deal and the flap becomes thicker. In the second stage, minute areas of slough, if present, are trimmed off as the margins are freshened.

In case the original opening is very large, or the postoperative complete cleft palate is very wide, with marked scarring, the closure may have to be accomplished in two stages. That is, the anterior part of the palate may be closed by delayed flaps from each side in the first stage. The aponeurosis of the palate is not freed at this time. Three months later the closure is completed by the two-stage Langenbeck operation.

In closing postoperative openings in the antrum following osteomyelitis of the jaw, the removal of malignant growths, or a Denker operation, the same procedure is employed. The flap is elevated from the inside of the check with the pedicle high and is then sutured back in place. At the same time the margins of the opening are freed. After a week, the flap is sutured in place around the opening. The pedicle may have to be cut in a week or ten days and replaced to the cheek, although this may not be necessary if the base is close to the opening.

The results of this method of closure of post-operative openings in the palate and antrum have been more satisfactory than any other method New has used. Cases of cleft palate which formerly were better taken care of by means of a plate, and cases in which mucoperiosteum is very thin, may be closed by the delayed flap.

Closure of Granulating Wounds with Reverdin-Halsted Grafts. KARL SCHAEPFER, Baltimore. *Bullletin of the Johns Hopkins Hospital*, April, 1923.

Every infected wound not suitable for secondary closure can be successfully covered with a very resistant, elastic skin by means of the Reverdin-Halsted transplantation. A thorough preliminary treatment with Dakin's solution, persisted in until relative sterility is obtained, is followed by grafting with small, deep grafts. This method is the technic of choice in treating defects following extensive laceration of the soft parts with secondary necrosis and suppuration, the grooves resulting from sequestrotomy for chronic osteomyelitis, and some cases with granulating areas following unsuccessful plastic operation for empyema. This method has also proved effective in the treatment of *x*-ray burns and of trophic non-specific ulcers of long standing that have proven refractory to all other measures.

Acetonemia-Acidosis-Alkalosis. JOHN PURL PARSONS, Ann Arbor, Mich. *The Journal of the Michigan State Medical Society*, May, 1923.

Acidosis and alkalosis are disturbances in the acid base balance, but in opposite directions. Clinically the symptoms are the same excepting that tetany frequently occurs in alkalosis.

The treatment of acidosis should be prophylactic. Soda should be given in small doses by mouth. In this way a serious acidosis as well as the danger of inducing an alkalosis may be prevented.

Acetonemia means an abnormal amount of acetone bodies in the blood. Treatment is to give glucose which is antiketogenic. To mild cases give it by mouth or rectum in form of five per cent solution. In cases of acetone intoxication give 20 per cent solution intravenously. Glucose, of course, is contraindicated when the carbohydrate metabolism is at fault as it is in diabetes.

In all cases water is essential as it is the medium through which all the exchanges take place.

There seems to be a condition induced by the factors at work producing acetone bodies in the organism which may be called acetone intoxication in contradistinction to true acidoses.

Surgical Treatment of the Ureter in Tuberculosis of the Kidney. WALTMAN WALTERS, Rochester, Minn. *Minnesota Medicine*, May, 1923.

Of the various methods employed at the Mayo Clinic for dealing with the ureter in nephrectomy for primary renal tuberculosis, the method of compression of the stem of the ureter, ligation with catgut and sterilization of the cut end by the cautery, produces the best end-results as measured by primary wound healing.

Nephrolithiasis as a Complication of Pregnancy. AIMÉ PAUL HEINECK, Chicago, Ill. *Medical Times*, April, 1923.

Nephrolithiasis in pregnant women determines the same anatomical changes in the affected kidney as are found in the calculous kidney of the non-pregnant. These stuctural changes manifest themselves by exudative, proliferating and degenerative processes, often suppurative in character; rarely, neoplastic. Under the influence of stones, the epithelium of the renal tubules may necrose, form cysts or become malignant. In the absence of infection, the lesions presented are those of atrophic and diffuse nephritis. Generally, the aseptic stage of calculus is brief. Infection, sooner or later, supervenes and gives rise to one or more of the following conditions: Pyelonephritis with or without abscess formation, perinephritis, sclerotic or suppurative pyonephrosis, and partial or complete conversion of the kidney into a sclero-lipomatous mass.

In every case, there should be a complete roentgenographic exposure of the bladder, both kidneys and ureters. Pyelography should be employed only when the dangers attending its use are far outweighed by the information which it offers. It enables us to determine the presence or absence of pathogic changes in the renopelvic outline and whether a doubtful shadow is or is not extra-renal.

If the symptoms be mild, obscure, indefinite and identifi-

cation of the x-ray shadows uncertain, the medical treatment of renal lithiasis is to be instituted and the pregnancy is permitted to proceed undisturbed. After the puerperium, or better, after the period of lactation, operative treatment is to be urged in suitable cases.

In the absence of contraindications, operation can be performed up to the sixth month of pregnancy, with safety to both mother and child. Up to that time in the absence of contraindications operative treatment is to be instituted in cases:

(a) of renal calculi too large to be spontaneously expelled through the natural channels. Even if the symptoms be recent in origin, or slight in intensity, nothing is gained by delay. Be not too optimistic as to small renal calculi; of all calculi, they are the ones most apt to lodge and become impacted in the ureter.

(b) of multiple calculi, irrespective of site, size, or number.

(c) of recuring attacks of excessive pain localized to, or radiating from, the renal region.

(d) of profuse or continuous hematuria.

(e) of acute pyelonephritis, of cortical or pericortical pus formation and pus collection, and bilateral pyelonephritis.

(f) of a calculus giving rise to urinary retention within the renal pelvis. A stone in the cortex of the kidney does not furnish as urgent an indication for operation as one in the renal pelvis.

(g) of obstructive calculous anuria. This condition calls for immediate relief.

(h) of bilateral nephrolithiasis. In bilateral cases, first operate on the least involved kidney provided there is no acute pathological process in the other kidney, requiring immediate attention. In from two to three weeks operate on the other kidney. If one of the kidneys be the seat of acute complications, it should be the first to receive surgical relief.

Experiences With Nonsurgical Biliary Drainage. EDWARD HOLLANDER, New York. *The American Journal of the Medical Sciences,* April, 1923.

Criticisms of the test when correlated with the known facts of experimental physiology are incorrect deductions from observations, rather than true objections to the fundamental principles of the reaction.

The diagnosis may be made by the correlation of the following observations:

1. Failure to obtain bile after successive stimulations indicates occlusion of the common duct.

2. The volume of "A" bile is dependent on the time interval between the injection and the gall-bladder response, as well as on the degree of dilatation of the common duct.

3. Failure to obtain "B" bile in *repeated* tests in indicative of an organic condition which is interfering with the function of the gall-bladder. This, in conjunction with the clinical picture, is a distinct indication for surgical intervention.

4. Sand or agminated crystals, is evidence of a tendency toward or of the actual presence of stones.

5. Swarming or colonizing bacteria indicate active infection.

6. Increased number of or massed leukocytes is indicative of an acute or subacute inflammatory reaction. The use of 2 per cent acetic acid brings out the nuclei more clearly.

7. Darker colors of "B" bile, clear to slightly turbid, (deeper shades of amber to tarry black), with sluggish or intermittent discharge, yielding more than 90 cc or less than 20 cc, are indicative of various degrees of gall-bladder stasis.

8. Columnar cells, especially tall columnar cells, usually degenerating, deeply bile stained, and in fan or rosette formation, when present *only* in the "B" *fraction*, represent catarrhal desquamation from the gall-bladder. Frequently intensively bile-stained detritus is also present.

9. Mild gall-bladder colic soon (and in some cases a few hours later), after the injections, indicates an inflammatory condition of the gall-bladder.

10. When gross infection (direct microscopical evidence) is absent, cultural methods are not reliable.

A light-colored bile may show marked microscopical evidence of disease, whereas a dark bile may show nothing more than its increased color concentration. The gall-bladder, in disease, may even lose its property of concentrating bile.

Four hundred and fifteen successful intubations were performed in 128 patients, chiefly gall-bladder suspects.

Operation was performed on 17 patients. Operation was advised in 11 patients solely on the evidence of the fractional bile examinations. In these 11 patients either the gall-bladder condition was merely suspected, or an incorrect clinical diagnosis had been made. In 8 of the patients, "B" bile was not obtainable after several attempts, and the operative findings disclosed sufficient gall-bladder disease to account for the failure of the gall-bladder response. In the remaining 9 patients, the "B" bile and the gall-bladder contents at operation corresponded both in color and microscopical findings. In 7 cases of this group, six to sixteen weeks after the cholecystectomy "B" bile could not longer be obtained.

Because of its bland action on the duodenal mucosa, 5 per cent peptone is suggested for diagnosis in preference to strongly hypertonic magnesium sulphate solutions.

Attention is drawn to the condition of "ampullospasm" in relation to gall-bladder stasis.

A normal reaction may be present in chronic cholecystitis. However, in most cases of gall-bladder disease we observe either failure of gall-bladder response, or microscopical abnormalities, or evidence of stasis. The correlation of these findings with other clinical data is essential to a correct diagnosis.

The practicability of the test is presented in the 17 operated cases. In many cases, the definite diagnosis of gall-bladder disease cannot be made by any other method.

Book Reviews

Montaigne and Medicine. By JAMES SPOTTISWOODS TAYLOR, M.D., F.A.C.S.; Commander, Medical Corps, U. S. Navy; Member Socéité Francaise d'Histoire de la Medécine. Octavo; 244 pages. New York: PAUL B. HOEBER, 1923.

Montaigne has always had a peculiar appeal for medical men. A sufferer from disease for a large part of his life, he had many contacts with medical science in all its phases. He was a close student of human nature and of mediaeval medical lore. His observations on medicine, written with a winning frankness and with a mind absolutely free from prejudices make delightful reading. As Dr. Taylor states, Montaigne had the true scientific mind. He never spoke with finality on any subject and he was always open to conviction. In this book, Dr. Taylor has collected the most interesting sayings of Montaigne on medicine and allied topics. It is, however, more than a collection. Dr. Taylor's own comments have a real charm and felicity of their own and are expressed in the style of a true literary artist.

The Vaso-Motor System. By SIR WILLIAM M. BAYLISS, M.A., LL..D., F.R.S., Professor General Physiology in University College, London. Octavo; 163 pages; illustrated. London: LONGMANS, GREEN & Co., 1923.

In this monograph, Bayliss discusses the various factors that produce changes in the diameter of the bloodvessels. In separate chapters he discusses the anatomy and property of bloodvessels, the effects of stimulation (chemical and mechanical), the vasomotor reflexes and chemical and pharmacological action on arterioles, the capillaries and the veins. Finally, the physiology of hemorrhage and shock are briefly outlined. The text is didactic in nature, not too complicated for the average medical man to understand and is written by a master in exposition. In these days, when physiology is coming into its own, this book is most timely and important.

Dosierungstabelle für Die Röntgen-Therapie. Von DR. G. HOLZKNECHT, Professor für Medizinische Radiologie and der Universität, Wien. Duodecimo; 33 zeite. Leipzig und Wien: FRANZ DEUTIKE, 1922.

This is a small pamphlet giving the various dosages employed in *x*-ray therapeutics at the present day. It should be of value to those interested in this subject.